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ARIZONA MEDICINE

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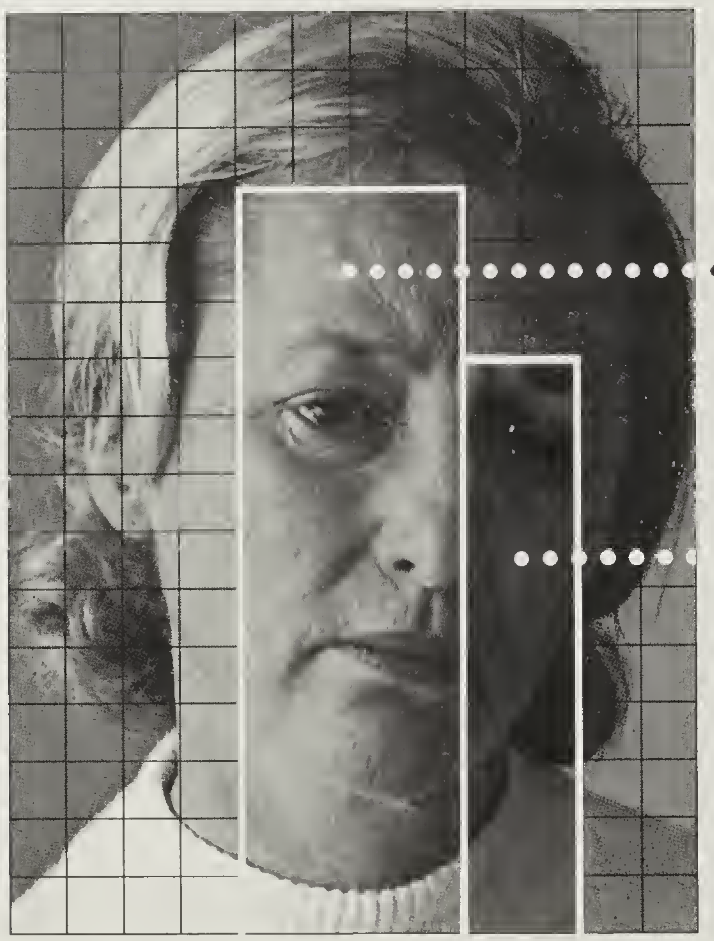
Predominant psychoneurotic anxiety
Associated depressive symptoms

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful

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According to her major symptoms, she is a psychoneurotic patient with severe anxiety. But according to the description she gives of her feelings, part of the problem may sound like depression. This is because her problem, although primarily one of excessive anxiety, is often accompanied by depressive symptomatology. Valium (diazepam) can provide relief for both—as the excessive anxiety is relieved, the depressive symptoms associated with it are also often relieved.

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anxiety states
with associated
depressive symptoms

surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate ts action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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ARIZONA MEDICINE

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Testing in Humans: Who, Where & When.

the weight of ethical opinion:

Few would disagree that the effectiveness and safety of any therapeutic agent or device must be determined through clinical research.

But now the *practice* of clinical research is under appraisal by Congress, the press and the general public. Who shall administer it? On whom are the products to be tested? Under what circumstances? And how shall results be evaluated and utilized?

The Pharmaceutical Manufacturers Association represents firms that are significantly engaged in the discovery and development of new medicines, medical devices and diagnostic products. Clinical research is essential to their efforts. Consequently, PMA formulated positions which it submitted on July 11, 1975, to the Subcommittee on Health of the Senate Labor and Public Welfare Committee, as its official policy recommendations. Here are the essentials of PMA's current thinking in this vital area.

1. PMA supports the mandate and mission of the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research and offers to establish a special committee composed of experts of appropriate disciplines familiar with the industry's research methodology to volunteer its service to the Commission.

2. PMA supports the formation of an independent, expert, broadly based and representative panel to assess the current state of drug innovation and the impact upon it of existing laws, regulations and procedures.

3. When FDA proposes regulations, it should prepare and publish in the *Federal Register* a detailed statement assessing the impact of those regulations on drug and device innovation.

4. PMA proposes that an appropriately qualified medical organization be encouraged to undertake a comprehensive study of the optimum roles and responsibilities of the sponsor and physician when company-sponsored clinical research is performed by independent clinical investigators.

5. PMA recognizes that the physician-investigator has, and should have, the ultimate responsibility for deciding the substance and form of the informed consent to be obtained. However, PMA recommends that the sponsor of the experiment aid the investigator in discharging this important responsibility by providing (1) a document detailing the investigator's responsibilities under FDA regulations with regard to patient consent, and (2) a written description of the relevant facts about the investigational item to be studied, in comprehensible lay language.

6. In the case of children, the sponsor must require that informed consent be obtained from a legally appropriate representative of the participant. Voluntary consent of an older child, who may be capable of understanding, in addition to that of a parent, guardian or other legally responsible person, is advisable. Safety of the drug or device shall have been assessed in adult populations prior to use in children.

7. PMA endorses the general principle that, in the case of the mentally infirm, consent should be sought from both an understanding subject and from a parent or guardian, or in their absence, another legally responsible person.

8. Pharmaceutical manufacturers sponsoring investigations in prisons must take all reasonable care to assure that the facilities and personnel used in the conduct of the investigations are suitable for the protection of participants, and for the avoidance of coercion, with a respect for basic humanitarian principles.

9. Sponsors intending to conduct nontherapeutic clinical trials through the participation of employee volunteers should expand the membership and scope of its existing Medical Research Committee, or establish such an internal Medical Research Committee, with responsibility to approve the consent forms of all volunteers, designs, protocols and the scope of the trial. The Committee should also bear responsibility to ensure full compliance with all procedures intended to protect employee volunteers' rights.

10. Where the sponsor obtains medical information or data on individuals, it shall be accorded the same confidential status as provided in codes of ethics governing health care professionals.

11. PMA and its member firms accept responsibility to aid and encourage appropriate follow-up of human subjects who have received investigational products that cause latent toxicity in animals or, during their use in clinical investigation, are found to cause unexpected and serious adverse effects.

12. PMA supports the exploration and development by its member companies of more systematic surveillance procedures for newly marketed products.

13. When a pharmaceutical manufacturer concludes, on the basis of early clinical trials of a basic new agent, that a new drug application is likely to be submitted, a proposed development plan accompanied by a summary of existing data, would be submitted to the FDA. Following a review of this submission, the FDA, and its Advisory Committee where appropriate, would meet with the sponsor to discuss the development plan. No *formal* FDA approval should be required at this stage. Rather, the emphasis should be on identification of potential problems and questions for the sponsor's further study and resolution as the program develops.

The PMA believes that health professionals as well as the public at large should be made aware of these 13 points in its Policy on Clinical Research. For these recommendations envisage constructive, cooperative action by industry, research institutions, the health professions and government to encourage creative and workable responses to issues involved in the clinical investigation of new products.

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1. For the relief of symptoms associated with cerebral vascular insufficiency.
2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.
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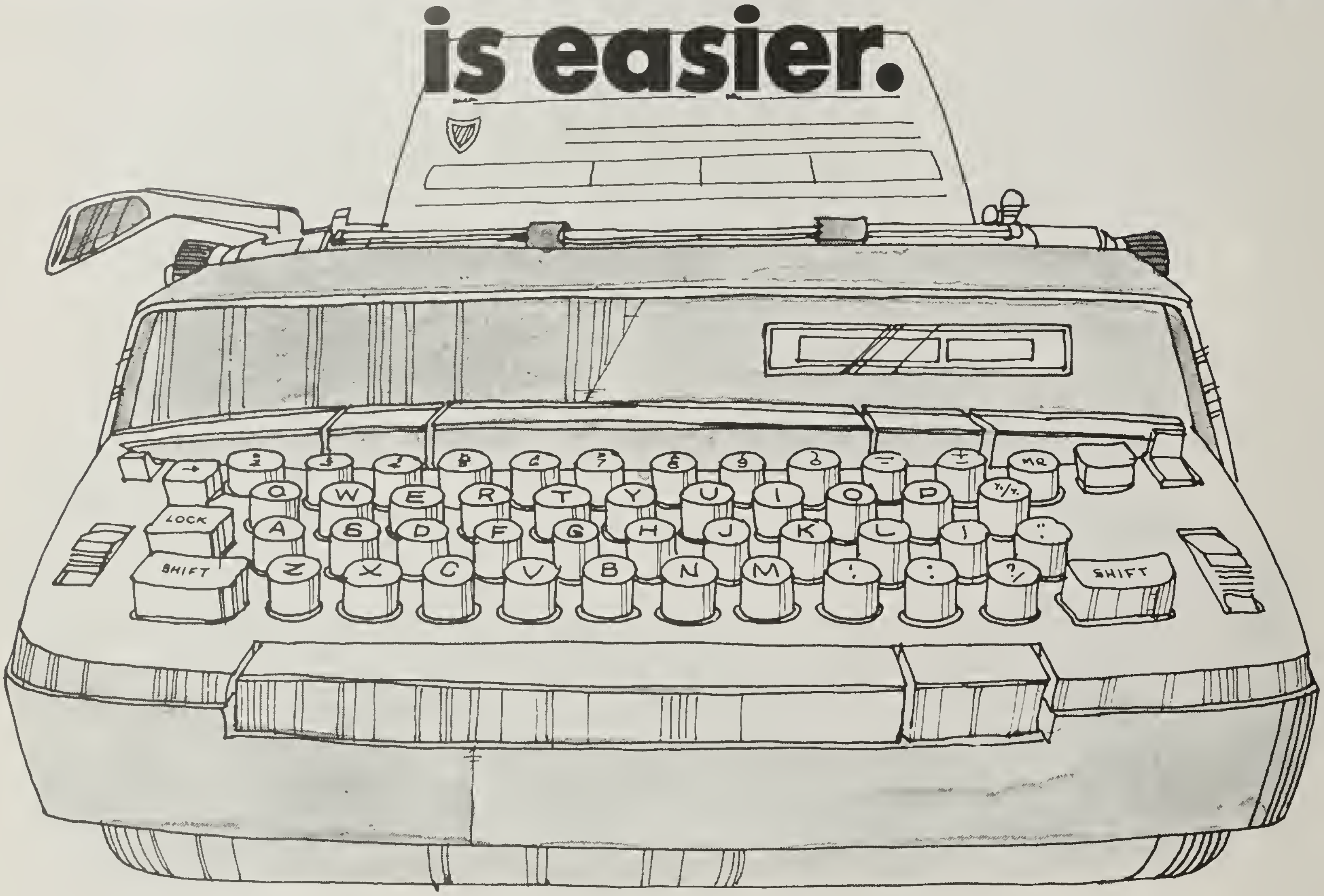

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Indications: For the relief of cerebral and peripheral ischemia associated with arterial spasm and myocardial ischemia complicated by arrhythmias.

Precaution: Use with caution in patients with glaucoma. Hepatic hypersensitivity has been reported with gastrointestinal symptoms, jaundice, eosinophilia and altered liver function tests. Discontinue medication if these occur.

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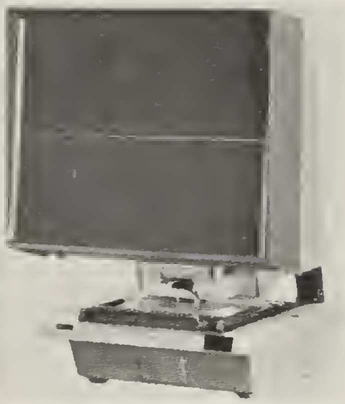

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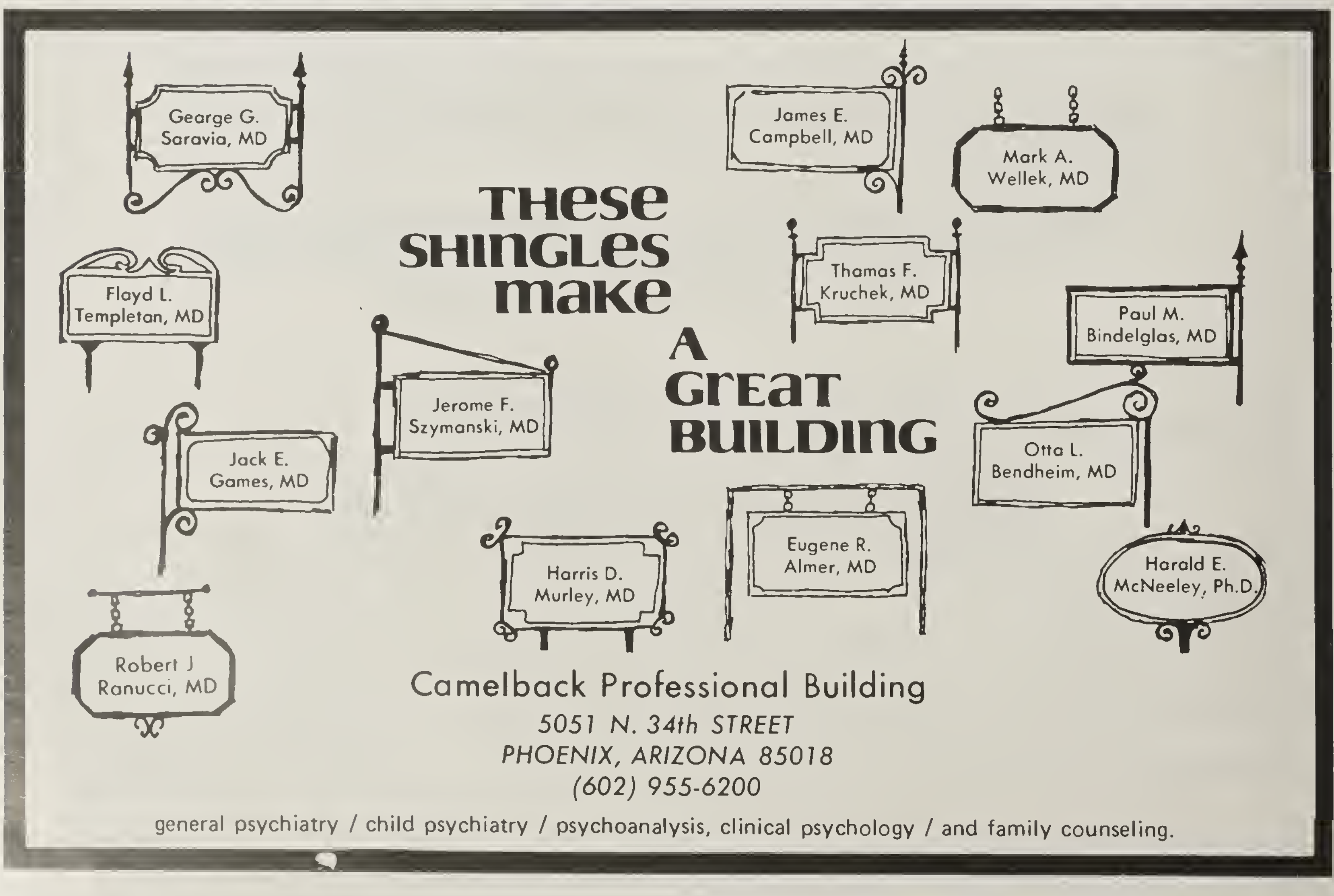

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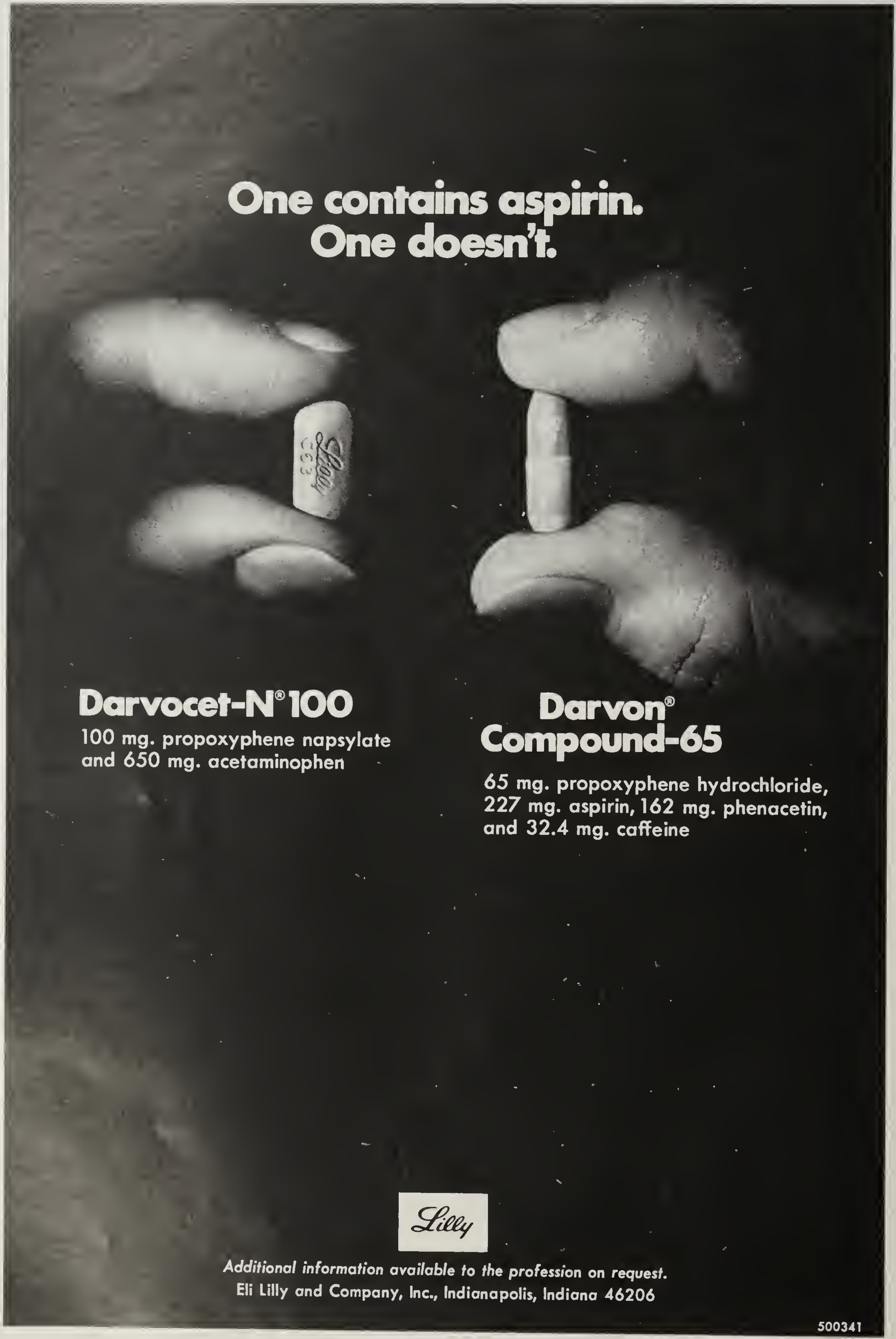

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Original Articles

HYPERINSULINISM DUE TO PANCREATIC ISLET CELL HYPERPLASIA REPORT OF A CASE IN AN ADULT

JOYCE R. BRADLEY, M.D.
JOHN P. HEILEMAN, M.D.

Wilder and co-workers first established the correlation between pancreatic islet cell neoplasm and clinical hyperinsulinism in 1927[1], but it remained for Graham[2], to remove the first functioning islet cell adenoma and effect a cure in 1929. Since that time the total number of cases reported approaches 1,000[1-11]. Approximately 80% of insulinomas are benign and single. In 10% metastases are evdient at the time of diagnosis and another 10% have multiple benign adenomas or islet cell hyperplasia[12]. Functioning islet cell tumors are rarely encountered in children but hyperinsulinism is occasionally found with hyperplasia of the islet cells in this age group; it is almost never seen in adults[9]. It is the purpose of this paper to present a confirmed case of clinical hyperinsulinism in the adult due to diffuse islet cell hyperplasia.

CASE REPORT:

A 48-year-old man was initially seen at the Veteran's Administration Hospital Clinic with a six month history of increased hunger, forgetfulness, confusion episodes and mental fugues. When initially examined he developed an episode of blank staring associated with disorientation and mild agitation. He was referred to another hospital where a blood sugar, in the emergency room, was 48 mg.%, and intravenous glucose administration promptly corrected his behavior. He was admitted to the hospital and had a brief work-up but was discharged without a definitive diagnosis. He once again presented to the V. A. Clinic with a blank staring episode, and was referred to St. Joseph's Hospital for further evaluation. The past history documented injury to the right lower extremity while in the Army in 1960, with subsequent multiple orthopedic and plastic reconstruction procedures but was otherwise essentially negative. He admitted to modest use of alcohol and tobacco. The family history was negative.

Physical exam on arrival in the emergency room revealed a well-nourished, medium-statured man who was somewhat confused, with normal vital signs. The positive findings included nystagmus on lateral gaze, myotic pupils with poor reaction to light, fine tremor of the extended hands, and poor performance on tests of coordination, finger-to-nose, knee-shin and past pointing undertaken with hesitancy and great difficulty. There was post-surgical absence of the right second, third and fourth toes with scarring of the dorsum of the right foot subsequent to previous reconstructive orthopedic surgery.

The blood sugar the following morning was 19 mg.%, and fasting blood sugar after overnight fast on the third hospital day was 27 mg.% associated with a plasma immunoreactive insulin level of 805 uU./ml. Serum electrolytes were normal and fasting and one-hour post-Cortrosyn plasma cortisol levels were 19 and 30 mg./100 ml. respectively. 24 hour urinary steroids revealed levels of 17-KS of 13.2 mg. and 17-KGS of 17-4 mg.; the 24-hour 5-HIAA excretion was 14 mg. The serum Dilantin level was unmeasurable, BSP was 4%, and liver enzymes were normal. The acid phosphatase was 1.1 IU. The VDRL was positive as was the FTA; subsequent spinal fluid serology obtained per lumbar puncture was negative. The EKG, chest x-ray, skull x-rays, brain scan, liver and spleen scan, IVP,

---

Reprint Requests: John P. Heileman, M.D., Endocrinology Associates, P.A., 3411 North Fifth Avenue, Phoenix, Arizona 85013.

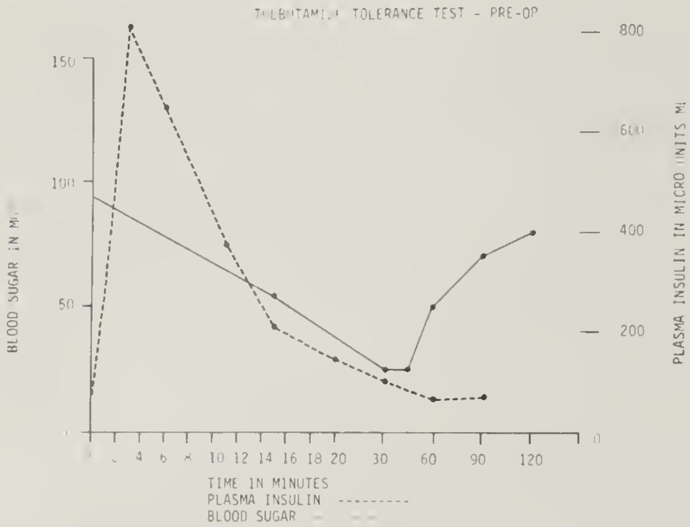

Figure 1: The response of plasma immunoreactive insulin and blood sugar to the intravenous injection of tolbutamide preoperatively in a patient with islet cell hyperplasia is demonstrated. The dash line represents plasma immunoreactive insulin and the solid line the blood sugar response.

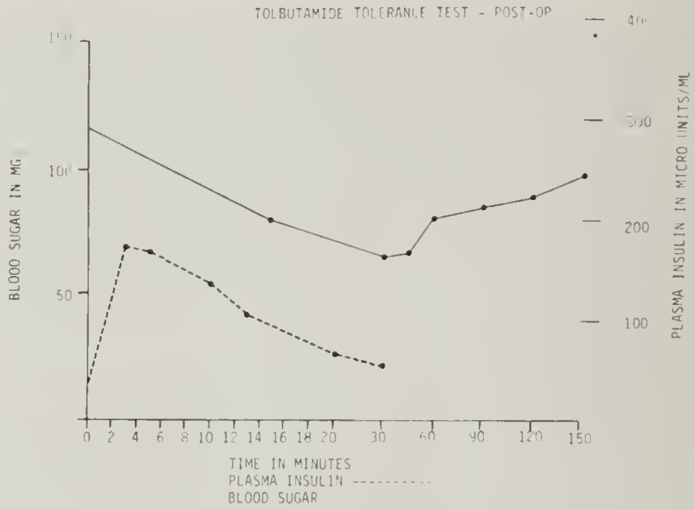

Figure 2: The response of blood sugar and plasma immunoreactive insulin following the intravenous administration of tolbutamide to a patient with islet cell hyperplasia after 95% of the pancreas was removed. The plasma insulin is indicated by the dash line and the blood sugar levels by the solid line.

upper GI series and barium enema were normal. An abdominal aortogram with celiac axis arteriogram failed to reveal the presence of a tumor.

During initial evaluation he was treated with a general diet, but during the periods of overnight fasting, he became extremely hungry and usually succeeded in convincing the night-duty hospital personnel to bring him additional feedings and sandwiches. In spite of this his blood sugars would repeatedly be less than 40 mg./100 ml. in the morning and on the third day he had a tonic seizure superimposed on his confusion episode. A tolbutamide tolerance test was subsequently undertaken with care (Figure 1). After appropriate penicillin therapy for the positive serology and following the documentation of grossly elevated plasma immunoreactive insulin levels associated with fasting hypoglycemia, surgery was recommended. At laparotomy no tumor was identified in the pancreas and a 95% pancreatectomy with choledochotomy and excision of multiple intra-abdominal lymph nodes was undertaken. Pathological examination of the tissues including microscopic examination revealed diffuse hyperplasia of the islets. Subsequent electron microscopy of the tissues documented the predominant cell type to be D-1 type.

Postoperatively transient episodes of hyperglycemia (with blood sugars in the 200 mg.% range) were noted. Two weeks post-surgery, a repeat tolbutamide test was undertaken after an overnight fast (Figure 2).

When seen in followup six weeks post-surgery the one-hour postprandial blood sugar was 111 mg.%. Some ten weeks post-surgery the patient was feeling well. He had experienced no recurrence of his "episodes." The fasting blood sugar

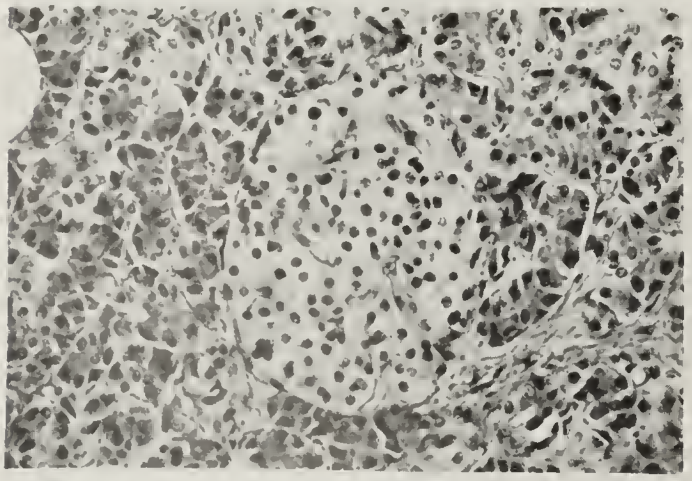

Figure 3: Normal islet cell morphology as seen with light microscopy.

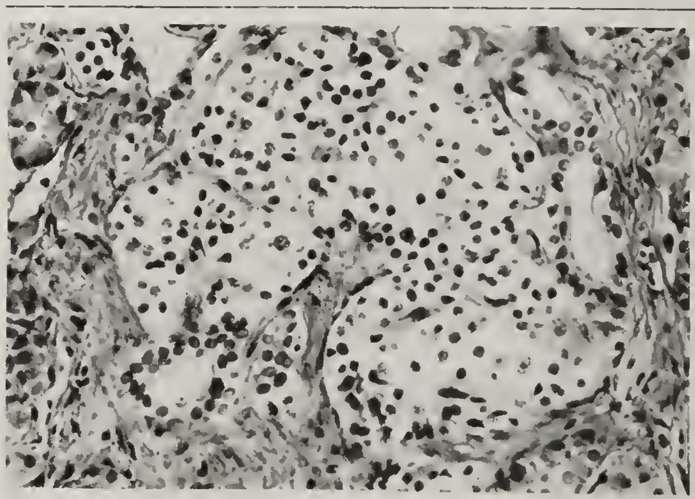

Figure 4: Islet cell hyperplasia from this patient as seen with light microscopy.

was 109 mg.%, and the fasting plasma insulin level 5.6 uU./ml.

The pathology slides were later reviewed by Dr. Stefan Fajans[1] of the University of Michigan and his staff pathologist, who confirmed findings of diffuse hyperplasia, with no evidence of capsule surrounding the individual islets, and no other evidence of microadenomatosis (Figures 3 and 4).

DISCUSSION:

In our patient we have documented the occurrence of fasting hypoglycemia associated with elevated levels of immunoreactive insulin which remitted following excision of 95% of the patient's pancreas. Histologically, islet cell hyperplasia was found and verified by several independent pathologists. A review of the world's literature indicated that this was an unusual circumstance.

In a large series of suspected insulinomas subjected to surgery and reported in several publications by Whipple and Frantz[8, 2] there were nine cases in which no tumor was found at operation; of these five had hyperplasia of the islets diagnosed pathologically, one had suspected hyperplasia. All of the patients with islet cell hyperplasia were very young, with the exception of a 50-year-old male, whose symptoms were not improved at surgery and who subsequently died by suicide two months postoperatively.

In the series reported by Dr. Longmire,[9] one patient, an infant with severe hypoglycemia, had hyperplasia and hypertrophy of the islet cells. The study reported by Howard[1] included a 47-year-old white male with hypoglycemic attacks (fasting blood sugar levels ranging between 30 and 70 mg.%), who had documented pancreatic hyperplasia at surgery, and in whom pancreatic resection was not elected; this patient later died during one of the attacks. Howard's summarization of the literature through 1950[1] included 11 cases of hyperinsulinism due to pancreatic hyperplasia, including his own case (above), a case reported by Virginia Frantz (above), and also included two children.

Hyperplasia of islets has been suggested as the cause of hypoglycemia in infants with hypersecretion of insulin. Nesidioblastosis may also produce hypoglycemia; this condition consists of new islet cell formation from pancreatic aciner cells.[6] In the neonate this condition is thought to be a hyperresponsiveness of islets to some stimulus in utero, possibly glucose or amino acids. Williams, Bryson and Hume report on a case of nesidioblastosis with marked hypoglycemia in a newborn.[6]

Ligation of the pancreatic exocrine ducts in dogs leads to diffuse hyperplasia of the islets, even in the presence of the atrophy of the pancreatic aciner cells. Brinck and Sponholz reported on a case of diffuse islet cell hyperplasia with hypoglycemic attacks in an adult with pancreatic lithiasis with obstruction of the pancreatic ducts,[11] a clinical situation comparable to ligation of the pancreatic ducts in the experimental animal.

Multicentric origin of benign and malignant tumors of the islet cells is suggested by the pathology observed in some series.[8] Islet cell hyperplasia may be part of the spectrum of this entity which is better termed pancreatic betacytoma.

In summary then, we have presented a patient who had fasting hypoglycemia associated with hyperinsulinism which was due to islet cell hyperplasia. The occurrence of this syndrome in an adult is indeed rare and requires careful preoperative investigation. Perhaps with a greater understanding of the interaction between the alpha, beta and delta cells of the islets of Langerhans a greater understanding of the inter-relationships and control of each cell's secretory products will accrue. This may help to explain in the future the occurrence of this pathologic condition.

ACKNOWLEDGMENT

We gratefully acknowledge the help of Stefan Fajans, M.D., Chairman, Division of Endocrinology and Metabolism, of the University of Michigan, who stimulated the writing of this report.

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[1] Personal communication.

THE CHROMOSOME 49, XXXXY SYNDROME

REPORT OF A CASE IN AN ADULT

MARGARET S. TENBRINCK, M.D., F.A.A.P.

JAMES H. BOYD, M.D.
SANDRA Y. BUCHIN, M.D., F.A.A.P.

Although 88 cases of the 49,XXXXY syndrome have been reported, most of these have been in children. The present report concerns a middle aged man who has been institutionalized since age 6. He has shown progressive deterioration, partly, at least, owing to the complications of his chromosome abnormality as well as to environmental deprivation. In addition he exhibited several features not previously described in this condition. He is the only known case in Arizona.

The first case of the 49,XXXXY syndrome was described in 1960 by Fraccaro[1] et al in a 7 year old boy. In the meantime, 87[2, 3] other cases have been reported. For the most part, these were first recognized in boys or young men with one notable exception,[4] a man of 35. The latter was not described in detail.

The cardinal triad of signs is mental retardation (usually severe), hypoplastic male genitalia, and proximal radio-ulnar synostosis. Other signs[5] include microcephaly, hypertelorism with upturned palpebral fissures ("mongoloid" slant), high arched or cleft palate, vertebral anomalies, clinodactyly of the fifth fingers, and coxa valga. Recent case reports by Karsh[3] describe two boys with congenital heart disease, making a total of 16 cases with this anomaly.

The present report concerns a middle aged man who has been institutionalized since age 6. It is intended as a longitudinal study of the deterioration which has occurred in a man with this anomaly. Furthermore, some additional physical signs have been observed in this patient which may enlarge the spectrum of abnormalities that can be associated with this condition.

REPORT OF A CASE

This 40 y/o caucasian man, was born to a then 25 year old mother and 31 year old father. Two years earlier, the mother had delivered a normal girl who is presently the mother of two normal children. The full term pregnancy was marked by persistent maternal nausea and vomiting. The mother states she gained very little weight, possibly 4.5 kilos (10 lbs.). She was under emotional strain during the gestation because of excessive alcoholic ingestion by her husband.

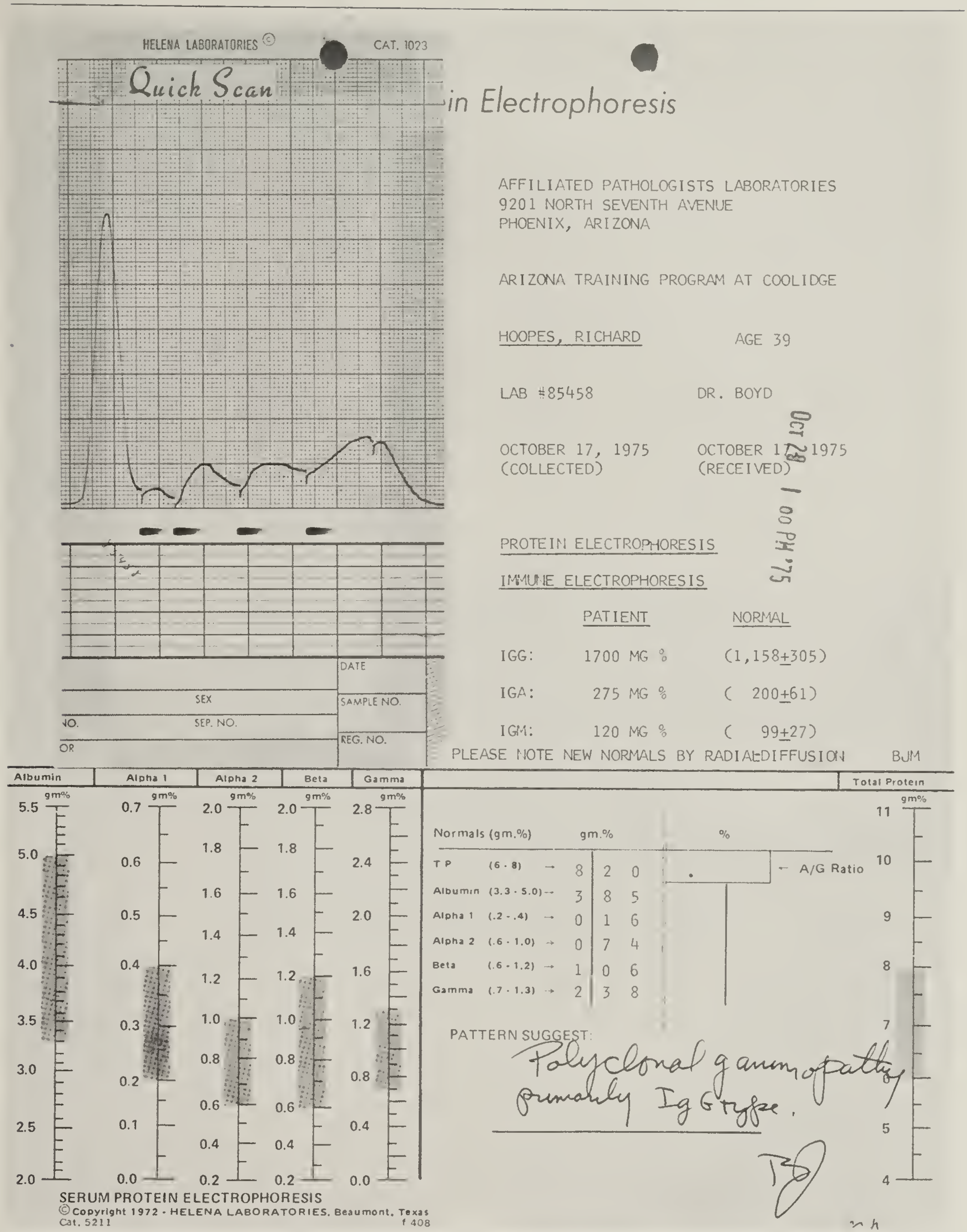
Aspirin was taken consistently because of presumed "arthritis" of the spine.

The delivery was in Mesa, Arizona, at a maternity home which shortly thereafter was closed and the records destroyed. She remembers that the baby weighed almost 2.25 kilos (5 lbs.) and was 50.75 cm. (20 in.) long at birth. She saw the child immediately and noted that he looked abnormal and had, specifically, a malformation of the right ear. He seemed weak, but his neonatal course was uneventful and he went home with his mother at 10 days, the customary length of hospitalization in 1936.

Shortly after the birth of the patient, the mother had a first trimester spontaneous abortion. A hysterectomy precluded any further pregnancies. There have been three known cases of twinning in the family. The maternal great-grandmother delivered a boy and girl, a maternal uncle's wife aborted twin girls, and a maternal aunt produced a boy and girl.

The early milestones of holding his head erect and noticing objects and voices did not seem delayed and compared favorably with his sister's progress. At 9 months he rolled over, cut his first tooth and started to creep. He sat unsupported at approximately one year. He imitated sounds, was able to stand alone, and drink from a cup at one year. He walked unattended at 2½ years, the same time he started to dress and undress himself, and make his wants known by speech. He is said to have been toilet trained near to one year of age.

From: Child Evaluation Center, Phoenix and Arizona Training Program, Coolidge. (Dr. Tenbrinck, F.A.A.P., Retired Dir., Child Evaluation Center); (Dr. Boyd, Staff Physician Arizona Training Program, (Coolidge 85228); Dr Buchin, F.A.A.P., Former Pediatrician, Child Evaluation Center, Phoenix.) Reprint requests to Dr. Boyd.



in Electrophoresis

AFFILIATED PATHOLOGISTS LABORATORIES
9201 NORTH SEVENTH AVENUE
PHOENIX, ARIZONA

ARIZONA TRAINING PROGRAM AT COOLIDGE

HOOPES, RICHARD AGE 39

LAB #85458 DR. BOYD

OCTOBER 17, 1975 (COLLECTED)
OCTOBER 17 1975 (RECEIVED)

OCT 28 1 00 PM '75

PROTEIN ELECTROPHORESIS

IMMUNE ELECTROPHORESIS

	PATIENT	NORMAL
IGG:	1700 MG %	(1,158±305)
IGA:	275 MG %	(200±61)
IGM:	120 MG %	(99±27)

PLEASE NOTE NEW NORMALS BY RADIAL DIFFUSION BJM

DATE
SEX
SAMPLE NO.
NO.
SEP. NO.
REG. NO.
OR

Normals (gm.%)		gm.%			%
T P	(6 - 8) →	8	2	0	. ← A/G Ratio
Albumin	(3.3 - 5.0) →	3	8	5	
Alpha 1	(.2 - .4) →	0	1	6	
Alpha 2	(.6 - 1.0) →	0	7	4	
Beta	(.6 - 1.2) →	1	0	6	
Gamma	(.7 - 1.3) →	2	3	8	

PATTERN SUGGEST: Polyclonal gammopathy primarily IgG type.

SERUM PROTEIN ELECTROPHORESIS
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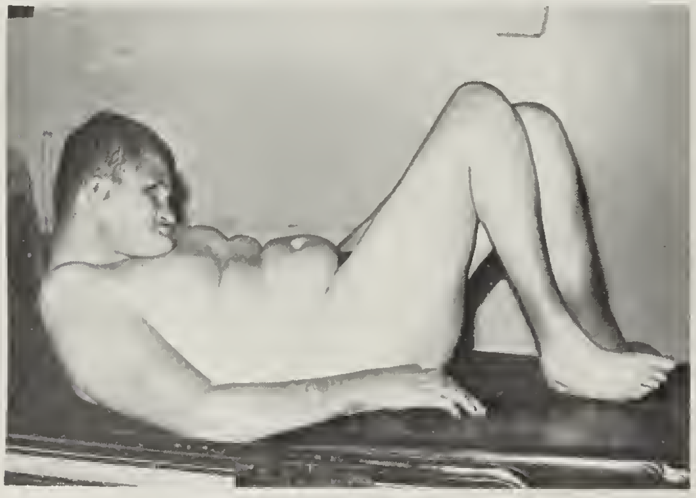
Cat. 5211 f 408

After breast feeding for four months, both mother and son contracted scarlet fever. He was extremely ill and in coma for four days. Thereafter he was fed formulas and the mother remembers him weighing 6 kilos (13 lbs.) at one year of age.

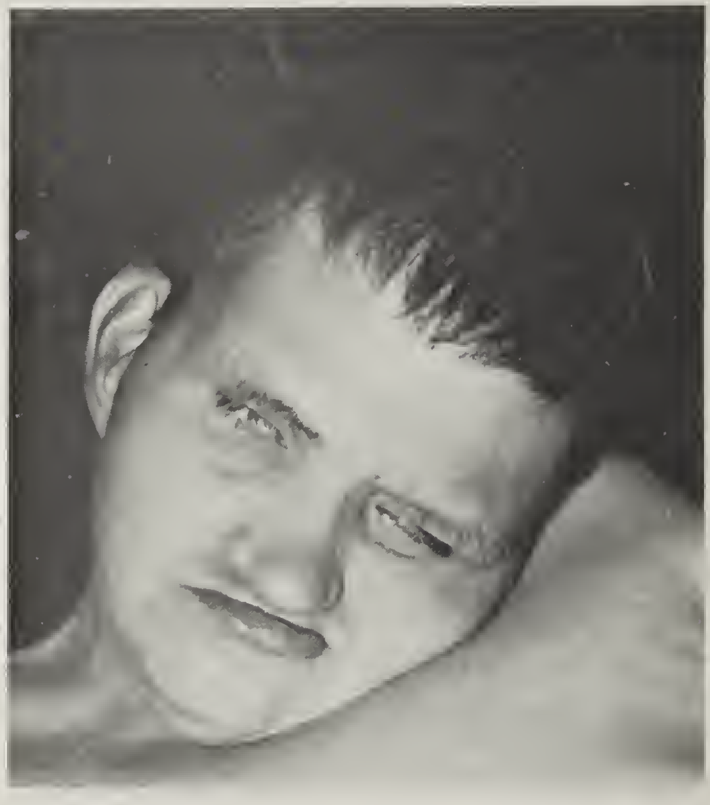
During his early childhood he was repeatedly ill with measles, rubella, mumps, and chickenpox. He did not move as fast as other children and did not play with them. He amused himself with toys and books. He had no apparent abnormal behavior. Because of the onset of centralized convulsions at age 4, he was examined by two physicians who concurred in the diagnosis of "mongolian idiocy."

At the age of 6, he was considered a danger to himself as he would run out into the street and, in general, had no appreciation of danger. He was admitted to the State Hospital where he remained for twelve years.

At the time of his admission to the State Hospital he did not talk. He had never attended any school and could neither read nor write. He appeared to have some interest in his surroundings. Height was 117 cm. (46 in.), or the 25th percentile; and weight 16.25 kg. (25 lbs.) below the 3rd percentile. He had "malformations" of both ears, the palate was high and arched. Both eyes demonstrated a convergent squint and bilateral lenticular opacities. The left elbow was enlarged without apparent deformity. Testes were small and located at lower end of the inguinal canals. He walked with a shuffling gait in which he spread his feet wide apart. Pes planus was noted. A satisfactory examination of his mental status was recorded as "impossible as he does not talk."



At the age of 12 the boy was seen in neurological consultation and diagnosed as a case of cerebral agenesis. Pneumoencephalography was not felt to be indicated. At 17 he developed bursitis in his left knee which became infected and was surgically drained. He apparently had a habit of walking on his knees. At times he showed affection and occasionally hit other patients more in a playful mood than as a sign of anger. He showed no temper tantrums, was quiet, and had no vocabulary.



At age 18 he was transferred to the Children's Colony at Coolidge, Arizona, with the diagnosis of possible Down's syndrome. Nine years later, on medical review, the possibility of Klinefelter or Fröhlich syndrome was entertained.

After admission to the Coolidge facility, he had multiple skin infections for which antibiotics have repeatedly been prescribed.

In 1973 he had a bout of bradycardia followed by atrial fibrillation which was treated with Digoxin. In 1974 an x-ray taken of his right arm showed chronic subluxation of the elbow. The last grand mal seizure was observed in 1974. He receives no anticonvulsant medication.

At the present time he has contractures and is unable to bear weight, thus it is impossible to obtain accurate weight and height measurements. When admitted to Coolidge in 1954 at age 18 he weighed 85 kilos (187 lbs.) and was 162.5 cm. (64 in.) tall. It is believed he weighs more now. The facial expression is vacant and he does not seem to be in contact with his environment. He makes no effort to communicate, but semes to respond minimally to those handling him.

The man is obese and eunichoid-appearing with girdle adiposity. The head circumference is 56 cm. His occiput is flattened. The brow is low with prominent orbitial ridges. He has microphthalmia with bilateral cataracts. In addition, he has a right corneal opacity which seems to preclude any vision in that eye. The left pupil

C
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M

CHILDRENS HOSPITAL
OF LOS ANGEL[illegible]

CHROMOSOME ANALYSIS REQUEST
AND REPORT

FLOOR/CLINIC DATE 1-28-74

HOO[illegible] RICHARD
2-2[illegible]6

Mrs. Edna Hoopes
6625 S. 7th Ave.
Phoenix, Ariz.

CHROMOSOME ANALYSIS 28

EXAMINATION:

100 ☐ CHROMOSOME ANALYSIS – STANDARD
101 ☐ CHROMOSOME ANALYSIS – AUTORADIOGRAPHY
102 ☐ CHROMOSOME ANALYSIS – FLUORESCENCE
103 ☐ CHROMOSOME ANALYSIS – OTHER
104 ☐ CHROMOSOME ANALYSIS – FAMILY MEMBER
105 ☐ CHROMOSOME ANALYSIS – BREAKAGE STUDY
106 ☐ BUCCAL SMEAR STUDY
107 ☐ PHYTOHEMAGGLUTININ STIMULATION TEST
108 ☐ TISSUE CULTURE – CHROMOSOMES
109 ☐ TISSUE CULTURE – OTHER

PHYSICIAN: Mx James H. Boyd, M.D. TELEPHONE: EXT.

ADDRESS: Arizona Training Program, Coolidge, Ariz. 85228

REFERRING DIAGNOSIS:

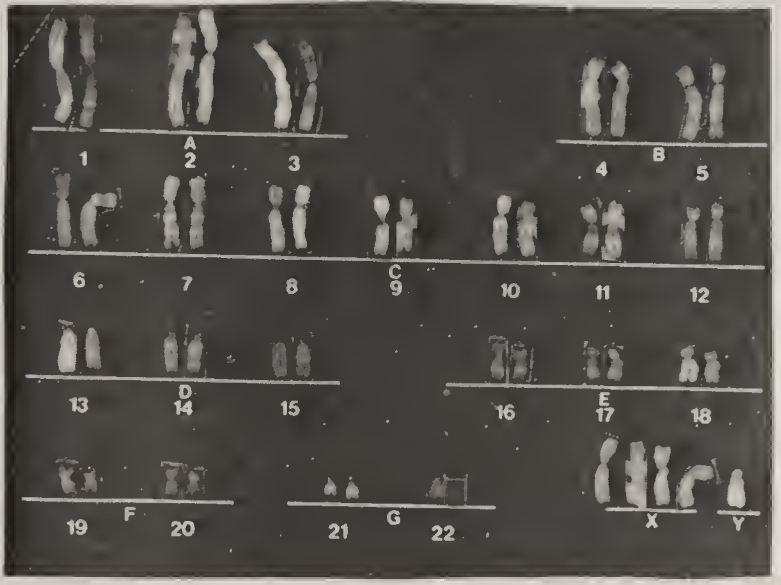
M. R., profound? mongoloid? "Marker 9"

HISTORY AND MAIN PHYSICAL FINDINGS:

March 20, 1974

CYTOGENETIC REPORT

NUMBER 74-99



RESULTS:
KARYOTYPE: 49,XXXXY

INTERPRETATION: A very abnormal karyotype.

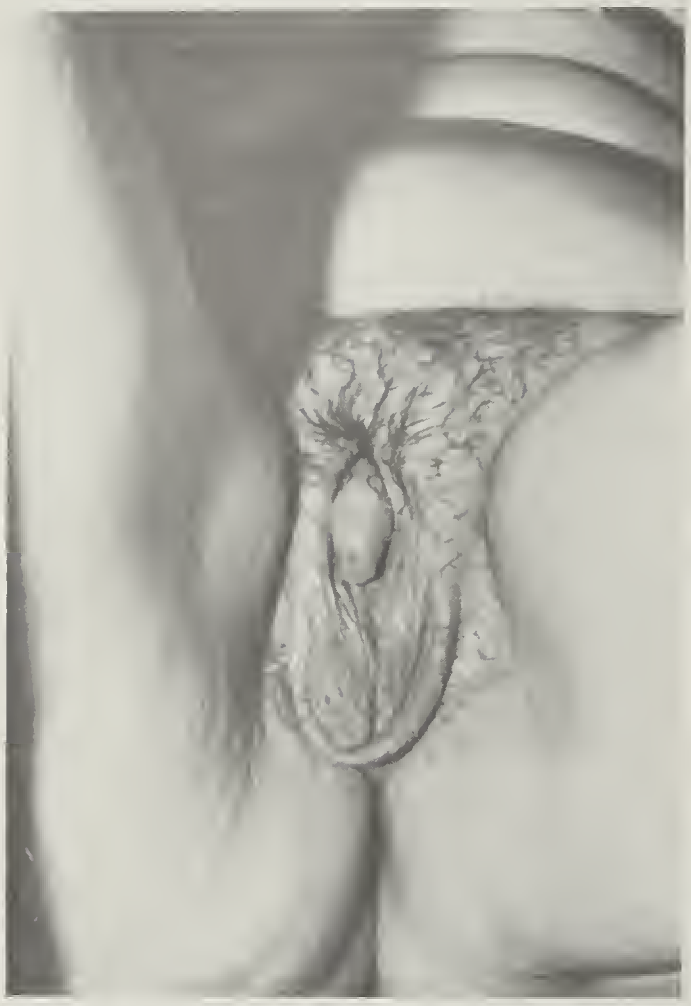
RECOMMENDATIONS: Request clinical description, a photo if possible, and the x-ray findings on bones.

O alfi
CYTOGENETICIST
Omar S. Alfi, M.D.

PATIENT CHART

CAT. 38-077 REV. 11/72

reacts to light. No mongoloid slant nor epicanthi were observed, as have been described in children[5]). The nasal bridge is depressed and nasal tip upturned and fleshy. The frenulum is short and lower lip protuberant. The palate has a flattened arch with evidence of a median raphe exostosis. A few front teeth have been extracted, the remaining incisors appear pegged. Prognathia (not micrognathia) is marked. Ears are lowset and there are several extra tabs on the right. The neck is short and wide with no frank webbing. He is unable to extend his neck and his



head remains flexed almost on his chest. Chest is short and wide with laterally placed nipples. He has fat pads in the breast areas. The thick chest wall caused the heart sounds to appear muffled. No masses or viscera were palpated in the abdomen. Pubic hair resembles a female escutcheon. The penis is small and short and the small scrotal sac is empty.

The abnormalities of the musculoskeletal system include proximally placed thumbs with subluxed metacarpol phalangeal joints. Although there are no simian lines, an almost total lack of palmar creases is noted. There is laxity of the proximal phalangeal joints. Fingers are spindly. There is an increased carrying angle of the arms resulting from the radial head dislocations. Hips and knees are held flexed. Pes planus is more marked on the left. There is considerable stasis dematitis over the left ibia. The dorsum of the left foot is edematous. The halluces are longer than the other toes and there is a space between toes one and two. Both knees are swollen. Sinus tracks are evident bilaterally resulting from abscesses. Deep tendon reflexes could not be obtained. Stroking the soles of his feet elicited withdrawal reactions. No cooperation could be obtained for any performance testing.

Laboratory Work

Cytogenetic report: 49,XXXXY (Courtesy of Childrens Hospital, Los Angeles)

Blood chemistry: Glucose 80 mg%, BUN 26 mg%, uric acid 4.5 mg%, calcium 8.9 mg%, inorganic phosphorus 2.9 mg% total protein 8.2 gm%, albumin 3.1 gm%, alkaline phosphatase 110 U/L, total bilirubin .26 mg%, cholesterol 161 mg%, SGOT 27 U/L, LDH 196 U/L, IGG 1700 mg%, IGA 275 mg%, IGM 120 mg%, IGE 400 U/ml.

Blood count: WBC 4.0 x 10^3 (segs 14, bands 6, eos. 7, baso 1, lymphs 58, monos 14), hct. 35%, hgb. 11.7 gm.

X-rays are, for the most part, unsatisfactory, as the patient is unable to straighten out or stand erect. He has severe kyphoscoliosis involving the cervical and thoracic spine. There is impingement on the intervertebral spaces, wedging of vertebral bodies, and considerable lipping. The heart is enlarged in transverse diameters. Elbows demonstrate subluxation and ankylosis. In the knees there is considerable irregularity of the joint surfaces, but no subluxation.

COMMENT

The first 49,XXXXY syndrome case was described fifteen years ago and since then 87 more cases have been added to the literature. The triad of signs in children is mental retardation, hypoplastic male genitalia and proximal radial synostosis.

We have had occasion to follow a 39 year old man who has been institutionalized since age 6. His speech has always been minimal. He reportedly stopped walking at age 13. He has demonstrated progressive physical and intellectual deterioration due, in part, undoubtedly, to environmental and social deprivation.

In addition, he has features not previously described in this condition which include: a normal head circumference, cataracts' and corneal opacity with microphthalmia, prognathia, abnormal nose and ears, eunichoid appearance, radial head dislocation, and severe flexion deformities of his vertebral column and extremities.

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SNAKEBITE IN ARIZONA

THOMAS W. BOYDEN, M.D.

The following article is timely inasmuch as the snakebite season is upon us. The author has accurately and concisely presented the views of the majority of physicians who have had the largest experience in treating snakebite. He points out that many victims are bitten, but not envenomized and that cryotherapy may further damage envenomated tissue. Emergency treatment by the cut and suction method is affirmed and outlined. The use of adequate amounts of antivenin is detailed with a caveat. Chelating agents are wisely not mentioned; steroids recommended only for non-specific purposes; and fasciotomy supported only in unusual cases. This reviewer believes that fasciotomy in sub-fascial deposition of venom preserves function, but venom is seldom injected this deeply. Dr. Boyden outlines minimum laboratory procedures.

The medical treatment of envenomation by poisonous animals has been a subject of fascination and confusion to physicians for many years. Medical practices have been dictated by tradition, anecdotes, fear and ignorance. In recent years clinical and laboratory information has transformed toxicology into a relatively precise body of knowledge which provides a scientific foundation for the rational therapy of human envenomation.

Arizona is abundant with poisonous animals and has been a leader in both numbers of envenomations and fatalities.

I. General Information

There are approximately 7,000 cases of snake envenomation reported in the United States each year, and countless other cases which are not reported.[1] During the past five years the number of deaths from snake venom poisoning has not exceeded 12 each year.[2] The family **Crotalidae** (rattlesnakes, copperheads, water mocassins) accounts for more than 90% of these bites, with children and snake handlers being the most frequent victims. In Arizona we have only two dangerously venomous snake groups: rattlesnakes and the coral snake. The coral snake genus in Arizona (**Micruroides**) is so diminutive in size as to present minimal danger to all but the small-

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From: Endocrinology Section, Dept. of Internal Medicine, Veterans Admin. Hospital, Tucson, AZ 85723 (Dr. Boyden).

est children.[3] There has been no recorded fatality from the bite of the Arizona coral snake. Some exotic venomous snakes are present in zoos and private collections. In the vast majority of snake envenomations in Arizona the rattlesnakes are the offenders.

A person presenting with the complaint of "snake bite" needs several considerations. There are many nonpoisonous snakes that bite and these wounds require little, if any, specific therapy. Snakebitten persons are usually extremely apprehensive, regardless of the kind of snake that did the biting. Exact species identification is often impossible by either the patient or the physician. It is incumbent on the physician to determine if the bite presented to him represents a harmless snake, and if not, to judge whether significant envenomation has occurred.

With the exception of imported reptiles, the Arizona physician must only decide whether a rattlesnake or a harmless snake is involved. If a rattlesnake is known to have caused the bite an assessment of the severity of envenomation must be made immediately. If the type of snake is in question the bite should be carefully inspected for the telltale one or two fang punctures in addition to the rows of pinprick-size tooth marks common to poisonous and nonpoisonous snakes alike.

Between 20-73% of documented rattlesnake bites result in no envenomation.[1, 2, 4, 5, 6] Finding fang puncture sites is not sufficient evidence to begin therapy. The earliest sign that envenomation has occurred is local swelling that begins immediately following the bite. The edema may progress proximally. Pain usually follows swelling and proximal lymphadenopathy often heralds systemic poisoning.

Systemic poisoning may be immediate or delayed several hours. The unpredictability of systemic illness, regardless of the severity of local damage, makes hospitalization necessary for all cases of known venomous snake poisoning. Anaphylaxis and death can occur within minutes of envenomation. Less immediate symptoms and signs of systemic envenomation include shock, diffuse intravascular coagulation,[7] hemorrhage from any site,[8] nausea and vomiting, bulbar paralysis, respiratory paralysis and pulmonary edema. The manifestations of systemic envenomation are often a combination of hemotoxic, neurotoxic and cardiotoxic, but any one of these may predominate.[9]

II. First Aid

Incision and suction of the fang puncture site is the treatment of choice if this can be done within the first hour after the bite. This can be lifesaving in the field or the emergency room. Up to 35% of experimentally injected venom can be retrieved if incision and suction is begun within several minutes, and some retrieval can be shown with a delay up to 30 minutes.[10] Gennaro and McCollough using rattlesnake venom tagged with radioactive iodine reported more than 50% recovery if incision and suction was started within 3 minutes of subcutaneous injection.[11] The incision need only be linear, not cruciate, no more than ½ inch in length and ⅛ to ¼ inch in depth, so that additional tissue injury from the incision itself is minimal. Suction should be continued for about 40 minutes. Time should not be wasted if telltale fang marks and signs of local envenomation (swelling, pain, discoloration) are present. If incision and suction are done promptly there is no indication for the application of a tourinquet.[10] Vascular occlusion concentrates the many toxic fractions in the extremity making local tissue damage more likely than if allowed to dilute slowly in the total body fluids. The overwhelming majority of snake envenomation victims recover without disability testifying to the body's ability to neutralize most toxins.

There is one exception to the non-use of a tourniquet: if envenomation appears very severe and medical supplies or facilities are hours away, a totally occlusive tourniquet may be applied to save a life by sacrificing the limb.

Icing the bite is dangerous (frostbite is a frequent sequelae) and of no proven therapeutic value.[11-14,17] Administration of antivenin has no place in first aid therapy out of medical facilities.

III. Hospital Care

All suspect poisonous snakebite cases should be observed in the hospital where frequent monitoring of vital signs, physical examination and laboratory testing should be done. If envenomation is apparent the patient should be skin tested with horse serum (0.02cc of a 1:10 solution) in anticipation of treatment with antivenin. If the skin test is negative this does not exclude possible anaphylaxis from the antivenin. If the skin test is positive densenisitization must begin early to allow use of antivenin later if necessary. The polyvalent antivenin available for use against all rattlesnake bites in the U.S. is a horse serum preparation made by Wyeth pharmaceuticals. Each vial, when reconstituted, is 10 milliliters.

Anticipation of complications in rattlesnake envenomation is of extreme importance. To that end an intravenous access should be started with a large gauge needle; blood should be typed and cross-matched on admission because this may become impossible at a time when systemic poisoning causes bleeding problems; a baseline blood count and bleeding profile, serum electrolytes, serum creatinine and liver function tests are invaluable reference points. Frequent checks on urine and stool for occult blood as well as electrocardiographic evaluation may all provide early clues of systemic disaster.

Polyvalent antivenin should be administered in all cases of systemic poisoning and in cases where local tissue damage in severe and progressive. This antivenin is the only specific antidote for all manifestations of rattlesnake envenomaton, and all other measures must be considered supportive. If antivenin therapy is judged necessary the initial dose should always be three or more ampules (10 ml each) initially, and additional amounts sufficient to halt the systemic or local manifestations for which it was started. Some adults have required 50 or more ampoules for severe envenomation. Probably the most common errors in antivenin therapy are underdosage or beginning therapy when it was not necessary. Remember approximately 50% of all bites result in no envenomation, don't be too anxious to treat. The harm in treating unnecessarily is the potential anaphylaxis and serum sickness attendant with administration of all non-human sera. Serum sickness will occur in approximately 75% of all patient receiving therapeutic amounts of antivenin. The onset is usually 7-10 days post-treatment. Antivenin should only be administered intravenously.

If either systemic poisoning or severe, progressive local envenomation is not present antivenin therapy is not indicated and simple expectant observation is all that is required. This group comprises well over 90% of all rattlesnake envenomation and these patients can be discharged to outpatient care within 24-48 hours. Proper wound healing should be monitored at weekly intervals. Antitetanus prophylaxis should be administered to all bitten persons when appropriate, and signs of infection monitored.

Systemic administration of glucocorticoids have no specific role in the treatment of snakebite.[15, 16, 17] Their use as nonspecific therapy in vascular collapse may be warranted but cannot be considered a substitute for antivenin. Steroids may actually decrease the effectiveness of circulating antibodies in neutralizing snake toxins.[18]

Since the natural history of most bites is complete recovery, cryotherapy and total excision of the bite must be considered inappropriate and potential causes of more limb disfigurement and dysfunction than the bite itself. Likewise, fasciotomy is an unnecessary procedure in all but the most unusual cases. This procedure is always associated with morbidity. The only indication for this surgical procedure is when local tissue edema is so severe as to compromise arterial circulation to the limb.

Bites from unusual or non-native snakes should always have the benefit of experienced expert consulation. Often type-specific antivenin must be obtained from centers located outside Arizona.

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DIAGNOSTIC ERRORS IN CLINICAL MEDICINE

PAUL J. RUTALA, M.D.

JAY W. SMITH, M.D.

These are interesting cases and they remind us again "every problem becomes very childish when once it is explained to you." (Author, Conan Doyle, Adventures of the Dancing Man). On a current topic, we can also appreciate how fertile is the malpractice field that we actively cultivate if our practice is without the "pursuit of excellence."

In any discipline with defined standards of excellence, deviations from those standards are inevitable. Such deviations must, however, be considered errors. In medicine, a profession from which both the lay public and the professionals themselves are demanding an ever-increasing level of competence, the potential for error and the ramifications thereof are constantly apparent to the clinician. The current stress on various care audit programs conducted within the medical profession dictates that errors need to be recognized promptly and remedied when possible.

Lack of knowledge is not a frequent cause of diagnostic error. Diagnostic error occurs more frequently because of fallibility on the part of clinicians in areas of data gathering, analysis and interpretation, or because of weighing information bits incorrectly. More specifically, mistakes in diagnosis are made because: 1) data are available but not gathered, 2) data are gathered but not analyzed, 3) data presented by other professionals are accepted without critical analysis, 4) data are noted but not synthesized appropriately, or 5) data are simply misinterpreted.

The following examples are mistakes made by numerous knowledgable physicians at a university hospital. These diagnostic errors do not stem from lack of knowledge, but from a lack of a methodical, disciplined approach to clinical problems.

CASE 1: Not Gathering Available Data. A 59 year-old woman was admitted to the university neurology service complaining of urinary incontinence, suprapubic colicky pain, anorexia, and a 20-pound weight loss. Physical examination revealed a large, firm, knobby liver. Neurologic examination recorded a normal gait but left lower extremity weakness and bilateral Babinski reflexes. A chest roentenogram showed multiple nodular densities throughout both lung fields. The initial impression was that the patient had a neoplastic spinal cord lesion; however, a myelogram was within normal limits, showing no evidence of obstruction, significant extradural defect, or cord widening.

On hospital day 5 the patient was transferred to the university medical oncology service with a presumptive diagnosis of widespread neoplasm of unknown origin. The first pelvic examination done on the 8th hospital day revealed a firm cervical mass which was a poorly differentiated epidermoid carcinoma.

The patient was treated with multiple antineoplastic agents but failed to respond favorably and died on the 17th hospital day. Autopsy disclosed extensive local tumor involvement (uterus, right ovary, right ureter, pelvic peritoneum, pelvic lymph nodes) and distant metastases (liver, both lungs, pulmonary lymph nodes) from her cervical carcinoma.

Comment: In this patient the correct diagnosis was made antemortem but on day 8 instead of day 1 as would have been readily possible. The patient's course would not likely have been

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From: U of A, College of Medicine, Arizona Medical Center, Tucson, AZ 85724. (Reprint requests to Dr. Smith.)

altered by earlier diagnosis, but in the interim between admission and diagnosis she did undergo testing which would have been unnecessary had the complete examination been performed upon admission. Further, in these cost-conscious days, the hospital bill was undoubtedly increased by the delay and unnecessary testing.

CASE 2: Not Analyzing Data Already Gathered. A 53 year-old man was transferred to the university medical service from a local hospital after a difficult course following a transurethral resection of the prostate. The surgical specimen had shown granulomatous prostatitis. On admission, physical examination disclosed a febrile patient with numerous draining skin lesions and purulent discharge from both ears. The chest roentgenogram revealed a cavitating consolidating infiltrate of the right upper lobe. The working diagnosis was Wegener's granulomatosis, based on the clinical picture and skin biopsy material.

On hospital day 3 the patient complained of pain in the left shoulder, elbow, and hand. An electrocardiogram suggested a previous strictly posterior infarction, i.e. a prominent R-wave in precordial lead V^1. On the 5th hospital day a recurrence of pain prompted another electrocardiogram which disclosed definite change: ST segment elevations were present in limb leads II, III, and a VF and ST segment depressions were seen in precordial leads V^2 and V^3. On the 6th day the patient was transferred from an isolation unit to the open ward. A nurse's note that day recorded the patient was complaining of "left shoulder and arm pain which was radiating down to the wrist." Vital signs were normal except for a pulse rate of 112 a minute; the patient was described as appearing in distress and slightly diaphoretic. The house officer was called and yet another electrocardiogram was taken, showing ST segment elevations in limb leads II, III and a VF and precordial leads V^2 through V^6. The patient died suddenly two hours after the third electrocardiogram. Autopsy material confirmed Wegener's graulomatosis as the primary diagnosis and acute myocardial infarction as the cause of death.

Comment: A cardiac origin for this patient's pain was suspected and appropriate diagnostic maneuvers were performed. All the requisite data were gathered. The physicians caring for this patient did not take time to look at those data. The time was available — 24 hours between the second electrocardiogram and the patient's death. Analysis of all data available was not methodical. A disciplined clinician ignores no information; neither because of lack of time nor because (as in this case) a primary diagnosis makes a secondary, but potentially serious, diagnosis seem less likely or less important.

CASE 3: Accepting Another's Data Rather Than Gathering One's Own Data. A 36 year-old woman was admitted to the university medical service complaining of one month of nausea and one week of epigastric pain associated with a single episode of "coffee-ground" emesis. The past medical history revealed a similar episode eight years previously which had been attributed to gallbladder disease although records of that work-up were not available. Two weeks prior to this admission she had undergone elective sterilization with a tubal coagulation performed in the university hospital under laparoscopic visualization.

On physical examination at her admission to the medical service she was found to have stool and naso-gastric aspirate both positive for occult blood. Epigastric tenderness was elicited on abdominal examination.

During a six day hospitalization there was no change in hemoglobin concentration, no recurrence of vomiting and a normal upper gastrointestinal roentgenographic series. The stools on all other occasions were negative for occult blood. She was discharged on antacids, sedatives, and chlorpromazine with the diagnosis still unclear.

One day after discharge she was seen at another local medical care facility for follow-up where she complained of amenorrhea and was found to have a midline pelvic mass. Pregnancy testing was positive and the diagnosis of intrauterine pregnancy, twelve weeks duration by size, was made.

Comment: The diagnosis of pregnancy to explain this patient's nausea and vomiting was not even considered during the hospitalization. It was noted that she had had the tubal coagulation and presumed that a pregnancy would have been appreciated. The single stool positive for occult blood was weighted as more relevant in this patient than performing a complete physical examination, i.e., performing the pelvic examination. The true diagnosis was blatantly missed.

CASE 4: Appropriately Noting Data But Inappropriately Synthesizing Them (Correct Diagnosis Not Considered). A 65 year-old man had been followed in the university hospital out-patient clinics for two problems: asthma and multiple myeloma. He was first seen for asthma August 1973 and for therapy required various bronchodilators, cromolyn sodium and finally alternate-day steroids. He entered the hospital January 1975 because of status asthmaticus and was discharged on daily steroids. An x-ray film of the chest was normal except for a primary Gohn complex. Multiple myeloma had been diagnosed February 1974 and he was treated with various chemotherapeutic agents as an out-patient until February 1975 when he developed malaise, fever and sweating. A roentgenographic film of the chest disclosed a right middle lobe infiltrate and prompted hospitalization on the medical service where fever, rales over the right middle lobe area, and a sputum smear showing polymorphonuclear leucocytes and Gram-positive cocci led to a diagnosis of pneumococcal pneumonia. Blood cultures were negative and the sputum culture was nondiagnostic. The patient was treated for pneumococcal pneumonia, became afebrile, and was discharged after 72 hours even though his malaise was still present; the chest roentgenogram was unchanged at the time of discharge. The patient was seen in the out-patient clinic twice after discharge. Penicillin was changed to cephalothin, the chest roentgenogram remained unaltered, and the patient continued to feel lethargic. He eventually sought medical attention elsewhere, was admitted to another hospital, and the diagnosis of pulmonary tuberculosis was made from sputum examinations.

Comment: Both the house officers and the attending staff diagnosed pneumococcal pneumonia because of its well known association with multiple myeloma.[1] Many clues to the correct diagnosis were present and noted – calcified hilar lymph nodes, a primary Gohn complex seen on two chest roentgenograms prior to the episode of pneumonia, daily steroid therapy, and the lack of a positive bacterial culture. However, the diagnostic label was assigned and the mental blinders were on. All of the diagnostic possibilities were not considered and there was no step-by-step analysis of the data. The initial diagnosis was not abandoned even when additional information gathered made the original assessment much less tenable.

CASE 5: Misinterpreting the Data (Correct Diagnosis Discarded): A 41 year-old white male from near the Mexican border entered the university hospital because of abdominal pain, fever, jaundice, and confusion. There was a past history of binge drinking of alcohol. The patient appeared acutely ill with fever and tachypnea. The liver was firm, tender, and 25 cm. in overall height. Neither asterixis nor the stigmata of chronic liver disease were detected. The SGOT was 1310 units (normal 4-20), SGPT 830 units (normal 3-21), alkaline phosphatase 87 units (normal 30-100), bilirubin 9.9/100 ml, albumin 2.1 gm/100ml, and the prothrombin time 20.1 seconds. No hepatitis associated antigen was detected. A liver scan was originally interpreted as showing a moderately enlarged liver with diffusely poor uptake of the isotope. Later, two filling defects were appreciated. The diagnosis of amebiasis was considered but thought not to be consistent with the evidence of such marked hepatic necrosis. The working diagnosis was severe hepatitis with submassive necrosis. He was treated with low protein diet, neomycin, magnesium citrate, and steroids. On hospital day 3 hypotension, oliguria and metabolic acidosis developed. A cardiac arrest occurred from which he was resuscitated only momentarily and death followed. Autopsy revealed amebic hepatitis with two amebic abscesses within the liver, one of which had ruptured into the peritioneal cavity.

Comments: Here the correct diagnosis was considered but discarded. The proper interpretation of the liver scan was not appreciated until late in the patient's course; massive hepaitc failure was thought to be inconsistent with amebic abscess of the liver. However, hepatic failure is rare but documented in hepatic amebiasis.[2] Clearly the data were misinterpreted and proper weight and attention therefore were not given to the information which was in retrospect the most useful diagnostically, i.e. large filling defects on liver scan in a patient from an area highly endemic for amebiasis.

DISCUSSION: All the physicians involved in the care of the above-described patients had the basic knowledge to diagnose each case correctly. The mistakes made in Cases 1 and 3 are easily remedied: All that is necessary is the self-discipline to do all that is required to obtain complete data relevant to the problem. A pelvic examination in the patient with a working diagnosis of disseminated malignancy when malignant tumors of the female genital tract account for 22% of all cancer in women,[3] and in a young female patient with an unclear history and unexplained nausea is clearly indicated and necessary. The common physician ploy: "Pelvic examination — deferred" cannot be accepted without asking the obvious questions: "Why is it deferred?," "Until when is it deferred?", and "Who will accept the responsibility to see it eventually is performed?"

The correct diagnosis in Case 2 simply required desire, interest and enthusiasm to review the data that were obtained. The clinician's response to chest pain is almost programmed and mechanical, but it does no good unless review of the electrocardiogram is equally programmed. This carries over to disciplined, systematic review of all data during ward or chart rounds. Surely the university hospital must (if any institution must) accept the responsibility to impress upon medical students and house staff the absolute necessity of such systematic review and its paramount importance in patient care.

The error in Case 4 was that of prematurely labeling the patient with a common diagnosis, and then not modifying that label as further information was gathered which pointed to another diagnosis which should not have been ignored in this clinical setting. This error, too, can be corrected by disciplined, systematic data review; taking the time for the methodical consideration of various diagnostic possibilities and taking the time for the methodical consideration of new data gathered each day during the patient's course. Some cynicism is not a bad trait in the clinician: He must question at each turn his initial assessments and be prepared to change those assessments as new data become available and as the clinical course would dictate. Students and house officers must develop an aversion to "snap" diagnoses even when those are emotionally and/or intellectually appealing.[4] Weed, in his approach to the problem oriented medical record, cautions us to define problems only at a level of refinement justifiable from all data gathered, a level which is consistent with the physician's understanding, no lower and no higher than that understanding.[5] It is no sin to carry a problem on a problem list which is not resolved to a diagnosis — if our data really do not permit a diagnosis. Perhaps in Case 4 if the problem had remained "Right middle lobe infiltrate" the physicians involved in care of that particular patient would have realized that the data they had obtained really allowed no higher resolution of the problem.

In Case 5 the appropriate diagnosis was considered but was dismissed due to: 1) a relatively low likelihood of amebic liver disease presenting in this manner and 2) an initial misinterpretation of the liver scan findings. This sort of mistake can only be corrected by the clinician's constantly being aware of his fallibility and potential for error, by meticulously examining and re-examining each bit of clinical information with regard to each diagnostic possibility (common and uncommon), and by always keeping an open and analytical approach to patient management. In dealing with the uncommon diagnosis the possibility of error is constant because the physician's logic is maximally stressed to weigh each bit of information for or against each likely and unlikely diagnostic possibility.

The layman has difficulty accepting the inevitability of mistakes. The clinician, on the other hand, knows that mistakes are always possible and are made to a lesser or greater degree by all practicing physicians. The professional knows that errors will be made; but he, too, should find it always difficult to accept those errors. He can accept them only when he has done all he can to maximize his own self-discipline and diagnostic acumen. Further, in order to accept error, he must be responsible through peer review and other audit programs for increasing the awareness of need for self-discipline and thoroughness among his fellow professionals.

We who care for patients must recognize our mistakes when we make them and should accept them as potentially maturing lessons in clinical decision-making: With each error we make or see, our clinical abilities, our analytical powers, our methodicalness and our capacity for logical interpretation must increase. We need to accept the challenge of continual self-reevaluation as well as evaluation of our peers to keep "unavoidable" diagnostic error in clinical medicine to a true minimum.

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Indications: In adults, urinary tract infections complicated by pain (primarily pyelonephritis, pyelitis and cystitis) due to susceptible organisms (usually *E. coli, Klebsiella-Aerobacter, Staphylococcus aureus, Proteus mirabilis,* and, less frequently, *Proteus vulgaris*) in the absence of obstructive uropathy or foreign bodies. **Note: Carefully coordinate *in vitro* sulfonamide sensitivity tests with bacteriologic and clinical response; add aminobenzoic acid to follow-up culture media. The increasing frequency of resistant organisms limits the usefulness of antibacterials including sulfonamides.** Measure sulfonamide blood levels as variations may occur; 20 mg/100 ml should be maximum total level.

Contraindications: Children below age 12; sulfonamide hypersensitivity; pregnancy at term and during nursing period; because Azo Gantanol contains phenazopyridine hydrochloride it is contraindicated in glomerulonephritis, severe hepatitis, uremia, and pyelonephritis of pregnancy with G.I. disturbances.

Warnings: Safety during pregnancy not established. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been reported and early clinical signs (sore throat, fever, pallor, purpura or jaundice) may indicate serious blood disorders. Frequent CBC and urinalysis with microscopic examination are recommended during sulfonamide therapy.

Precautions: Use cautiously in patients with impaired renal or hepatic function, severe allergy, bronchial asthma; in glucose-6-phosphate dehydrogenase-deficient individuals in whom dose-related hemolysis may occur. Maintain adequate fluid intake to prevent crystalluria and stone formation.

Adverse Reactions: *Blood dyscrasias* (agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia); *allergic reactions* (erythema multiforme, skin eruptions, Stevens-Johnson syndrome, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis); *G.I. reactions* (nausea, emesis, abdominal pains, hepatitis, diarrhea, anorexia, pancreatitis and stomatitis); *CNS reactions* (headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo and insomnia); *miscellaneous reactions* (drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon). Due to certain chemical similarities with some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia. Cross-sensitivity with these agents may exist.

Dosage: Azo Gantanol is intended for the acute, painful phase of urinary tract infections. *Usual adult dosage:* 2 Gm (4 tabs) initially, then 1 Gm (2 tabs) B.I.D. for up to 3 days. If pain persists, causes other than infection should be sought. After relief of pain has been obtained, continued treatment with Gantanol (sulfamethoxazole) may be considered.

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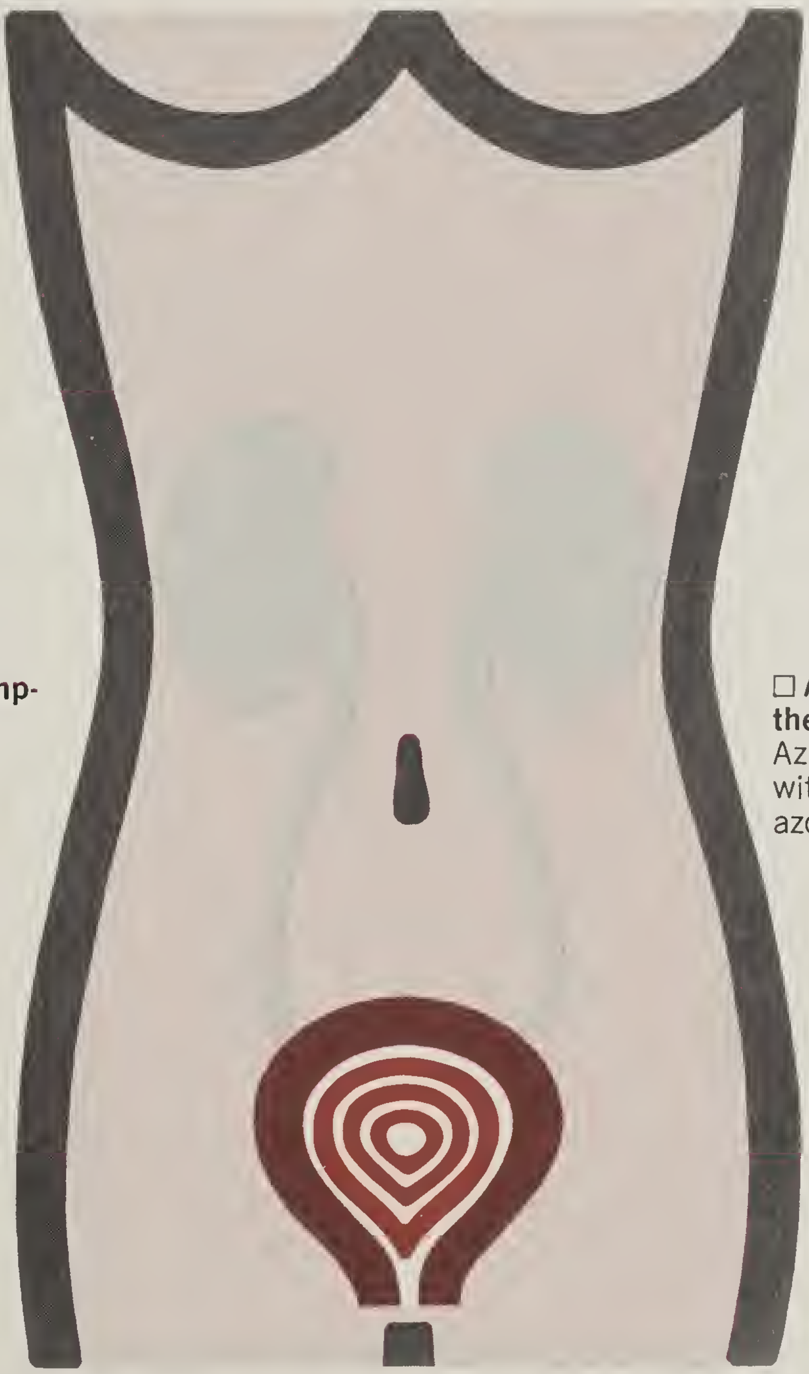

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Contraindications: Pre-existing elevated serum potassium. Hypersensitivity to either component. Continued use in progressive renal or hepatic dysfunction or developing hyperkalemia.

Warnings: **Do not use dietary potassium supplements or potassium salts unless hypokalemia develops or dietary potassium intake is markedly impaired.** Enteric-coated potassium salts may cause small bowel stenosis with or without ulceration. Hyperkalemia (>5.4 mEq/L) has been reported in 4% of patients under 60 years, in 12% of patients over 60 years, and in less than 8% of patients overall. Rarely, cases have been associated with cardiac irregularities. Accordingly, **check serum potassium during therapy, particularly in patients with suspected or confirmed renal insufficiency (e.g., elderly or diabetics). If hyperkalemia develops, substitute a thiazide alone. If spironolactone is used concomitantly with 'Dyazide', check serum potassium frequently**—both can cause potassium retention and sometimes hyperkalemia. Two deaths have been reported in patients on such combined therapy (in one, recommended dosage was exceeded; in the other, serum electrolytes were not properly monitored). Observe patients on 'Dyazide' regularly for possible blood dyscrasias, liver damage or other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving Dyrenium (triamterene, SK&F). Rarely, leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with the thiazides. Watch for signs of impending coma in acutely ill cirrhotics. Thiazides are reported to cross the placental barrier and appear in breast milk. This may result in fetal or neonatal hyperbilirubinemia, thrombocytopenia, altered carbohydrate metabolism and possibly other adverse reactions that have occurred in the adult. When used during pregnancy or in women who might bear children, weigh potential benefits against possible hazards to fetus.

Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in postsympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis; rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting (may indicate electrolyte imbalance), diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

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CONTRAINDICATIONS. Administration of Antivert (meclizine HCl) during pregnancy or to women who may become pregnant is contraindicated in view of the teratogenic effect of the drug in rats.

The administration of meclizine to pregnant rats during the 12-15 day of gestation has produced cleft palate in the offspring. Limited studies using doses of over 100 mg./kg./day in rabbits and 10 mg./kg./day in pigs and monkeys did not show cleft palate. Congeners of meclizine have caused cleft palate in species other than the rat.

Meclizine HCl is contraindicated in individuals who have shown a previous hypersensitivity to it.

WARNINGS. Since drowsiness may, on occasion, occur with use of this drug, patients should be warned of this possibility and cautioned against driving a car or operating dangerous machinery.

Usage in Children: Clinical studies establishing safety and effectiveness in children have not been done; therefore, usage is not recommended in the pediatric age group.

Usage in Pregnancy: See "Contraindications."

ADVERSE REACTIONS. Drowsiness, dry mouth and, on rare occasions, blurred vision have been reported.

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NATIONAL HEALTH PLANNING AND RESOURCES DEVELOPMENT ACT OF 1974 (P.L. 93-641)

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JOHN W. KENNEDY, M.D.

Ask your colleagues if she or he favors National Health Planning and you may be met with a blank stare or vehement denunciation of the Act. Perhaps we all lack overall knowledge of the goals or gaols, that await private medicine when the Act is fully mechanized.

Mrs. Marjorie Lynch, Undersecretary of HEW — 140,000 employees, 120 billion dollar budget is quoted as follows:

"Q. In your opinion, are such programs as medicare, medicaid and PL 93-641 forerunners of National Health Insurance?

A. No Question. We're already into a certain medical program. You can't say we don't have government medicine. The next step — taking all the people in the country, that's the major subject of debate." (Mrs. Lynch is parroted by all regional and local HEW officials that we have heard drone on this subject). (Journal AHA March 1976, P56).

H. Robert Cutheart, president of the American Hospital Association is quoted in their official journel "Our association . . . has shown superb leadership in advocating a progressive health plan for the nation."

"We have proposed and are still working very vigorously to promote *regional* Health Planning."

"We have promoted ambulatory care . . . (a noble cause) . . . and the involvement of the consumer in his own preventative and health maintenance programs. (Translation HMO's), and El Presidente further states "We shall want to devote more time to health maintenance programs." (Hospitals JAHA 1976).

The goals, as stated in the act, are somewhat like Norman the Conquerer devised for his newly conquered subjects in England He enrolled them in a Domsday Book.

So it is a goal of P.L. 93-641 to enroll all health service providers, regulate their charges, expansion, and standards of care.

This is done by the usual carrot and stick method. The local Health Planning Councils, made up of 60% consumers, begin inspecting and regulating, "to minimize the uneconomic disolution of health facilities and services," certificate of need."

Since the planning council has no "enforcement powers" it is up to the State Department of Health Services to "close down the hatch" if someone gets out if line. Finally HEW with all its money programs to the States simply begins to withhold funds if there is a continued non-compliance.

If you are not yet more confused you should be, so here is a final offering. There has been offered to the legislature, in New York State, a bill which would require the same demonstration of "a certificate of need" of any person to acquire and use a computerized transcipial scanner." The move is clear — next you may have to obtain a certificate of need for a new examining table!

So what can be done. It seems very important that physicians volunteer for Committee work where the council planning is done. Sure we are all to busy, hospital staff requirements and committee duties are overwhelming. But, your expertise and advise in the comprehensive health planning local committee is the only chance left to preserve our high standards of private medical practice and patient care.

Patient care these committees all understand, protecting your private practice is your concern, but the two are inseparable.

Remember the HEW Doomsday Book — P.L. 93-641.

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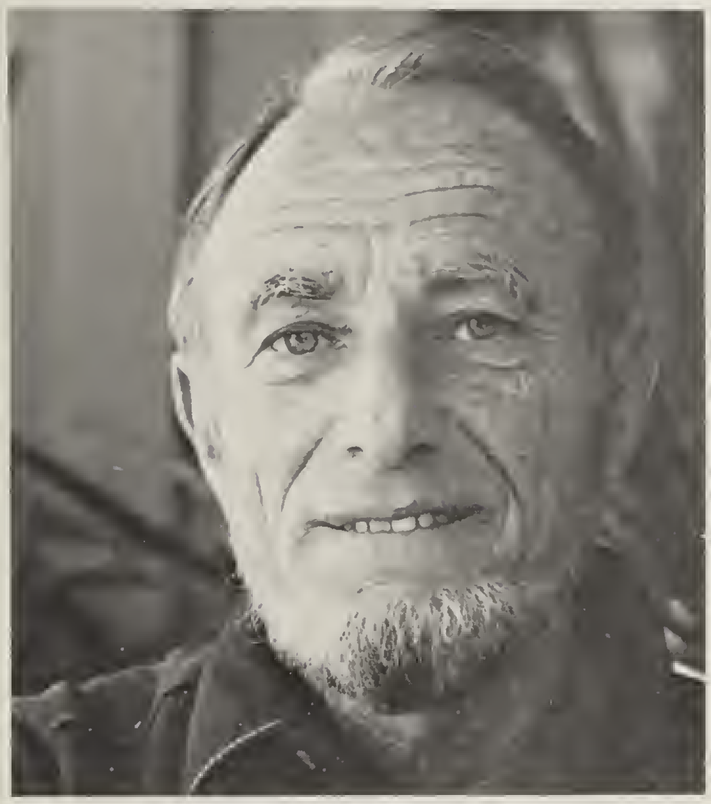
COMMUNICATION



EDWARD SATTENSPIEL, M.D.

Some 36 years ago, in 1940, the President of ArMA, D. F. Harbridge, M.D. commented in *Arizona Medicine* about the value of the First Annual Conference of Presidents and Secretaries of the County Societies convened by the State Medical Association. Possibly because of the impact of World War II, these conferences apparentry were not continued.

On May 22, 1976, the Presidents of all 14 County Medical Societies or their designated representatives were invited by me to join the Executive Committee and Staff of ArMA for a dinner meeting (with wives) at ArMA Headquarters. As a result, including the Executive Committee, there were gathered representatives of Apache, Coconino, Gila, Maricopa, Pima, Pinal, and Yuma counties.

This was a very informal and friendly get together, with no formal agenda. We talked about some of the things the State Association has been doing and is planning for the future. But, more importantly, we heard from all counties present something about their expectations, gratifications and disappointments in connection with our state activties. Because everyone was relaxed after good food and libation, the comments were open, honest, and, by and large, constructively critical.

I think we learned a lot about the "vital signs" of our membership around the state, and I hope we can be responsive to many of the problems and needs expressed. Subjects brought up included continuation of our efforts to find a more permanent solution to the malpractice problem, high fees charged by some specialists in urban areas and the cost of medical care in general, involvement of younger doctors in activities of organized medicine, and some of the programs of the American Medical Association.

Communication is the name of the game. My strongest reaction afterwards was that, although we may have some differences in attitudes around the state, the similarity of our problems, wherever we practice, is far more impressive. This may have been only the second annual meeting of County Society Officers at the State level, but I'm sure it won't be 36 more years until the third.

Dean's Page

MULTIPLE SCLEROSIS CLINICAL CENTER

NEAL A. VANSELOW, M.D.

Under terms of a new grant the Department of Neurology at the medical school has established a Multiple Sclerosis Clinical Center at University Hospital. This is the first clinical center in the country to be supported by the National Multiple Sclerosis Society. The $227,000 three-year grant provides funds for one additional faculty member in the department as well as nursing and technical support for a Multiple Sclerosis Clinic at Arizona Medical Center. The aim of the center is to follow approximately 120 patients with well-documented multiple sclerosis (MS) for a period from three to six years. Approximately 80 volunteers who do not have any neurological disease will be followed in a similar manner for comparison purposes.

At present there is no convincing laboratory model for MS; it seems probable, therefore, that the most meaningful research in MS in the future will somehow result from a study of patients with the disease. MS is still a disease of unknown cause and one for which no effective therapy exists. The clinic will examine patients in great detail four times a year on a routine basis and more frequently in the event of exacerbation in the disease. In addition, factors in the environment which may influence the natural history of the disease will be systematically explored with each patient at monthly intervals with the assistance of a trained research assistant, who is also a registered nurse. Blood, spinal fluid and tissue specimens will be stored and used both for local research and by investigators nationwide who are studying MS.

The project will also provide a facility for pilot study of potential therapeutic agents and facilities for collaborative research with research workers from other parts of the country who do not have access to adequate patient material.

It is estimated that there are approximately 500 patients with MS in the southern Arizona area. Close follow-up of house-bound and nursing home patients will allow the involvement of both patients and families in the research effort. It should also provide better symptomatic treatment and psychological support for both patients and families.

Dr. William A. Sibley, Head of the Department of Neurology, is Director of the Clinical Center and Dr. Colin Bamford is Director of the Multiple Sclerosis Clinic. Other personnel in the Department of Neurology actively engaged in the project include Dr. Jose Laguna, Assistant Professor of Neurology, and a nurse-research assistant, Mrs. Katherine Clark.

Initail efforts during the past few months have included tissue typing of all patients, which has confirmed findings in other centers that types HL-A3 and HL-A7 are considerably more common in the MS population; this suggests a genetically determined predeposition to the etiological agency, whether it be neuroallergy or chronic viral infection. Of considerable pertinence now is the Clinic's inability to find evidence that influenza vaccination has ever been associated with frank exacerbation of the disease in the past in any of its patients.

The University is gratified by the national recognition given to the Department of Neurology through the establishment of the Multiple Sclerosis Clinical Center and looks forward to future developments in this area.

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Seminars in Endocrinology and Metabolism

PANCREATIC ISLET CELL TUMORS: A BRIEF REVIEW

WOLFGANG SHEELY, M.D.

Continuing with the present issue of Arizona Medicine is the series of articles entitled "Seminars in Endocrinology and Metabolism." The purpose of these short review articles is twofold. First, due to the rapid proliferation of new knowledge in the field of endocrinology and the multiple tests available for their evaluation, short, clinically oriented reviews would enable the physician to keep abreast of these newer developments as they relate to their practice. In addition, with great stress being placed on voluntary recertification in many subspecialties, reviews such as they could serve as an authoritative, succinct teaching forum. The editors will endeavor to accomplish these goals by utilizing the talents of practicing physicians as guest contributors to this series. Feedback, both positive and negative, is encouraged in order to help us fulfill these objectives.

MARSHALL B. BLOCK, M.D. Editor

The islets of Langerhans may be the source of benign and malignant tumors some of which are associated with the remarkable propensity to produce hormonally mediated clinical syndromes. It therefore was thought instructive to review this type of tumor to bring into focus its many facets.

Pancreatic islet cell carcinoma is an uncommon tumor occurring in less than one person per hundred thousand population.[1] The clinical features of 52 patients with islet cell carcinoma were reviewed by Broder, e tal. Seventy-five percent of the patients had hormonally functioning tumors with the majority secreting insulin. The tumors were found primarily in the tail of the pancrea and were noted with equal frequency in men and women at a median age of 52 years. Metastases were mainly to the liver and by local extension. The most frequently reported presenting symptom was hypoglycemia, occurring in 90% of the functioning tumor patients. Nine percent were found to secrete both insulin and glucagon. Since glucagon is thought to come from the alpha-cells of the islets oversecretion can occur and mask hypoglycemia due to unopposed excessive insulin secretion. In contrast, pure glucagon secreting islet cell tumors have been described producing carbohydrate intolerance.[2] In the above series one patient had the Zollinger-Ellison syndrome while five others had peptic ulcer disease in association with hyperinsulinemia, however gastrin levels in these cases were not measured. Syndromes caused by excess adrenocorticotrophic hormone, melanocyte stimulating hormone, 5-hydoxyindolacetic acid, gastrin, insulin and glucagon, either alone or in combination have been reported.[3] An overall median survival of 908 days, from diagnosis to the last followup, was reported in one large series. However, malignant insulinomas may be slowly growing tumors and patients with such tumors have lived as long as 15 years.

Recent electron microscopic and biochemical studies have enhanced our understanding of how these hormonally producing cells function. For instance, the beta cell, as previously commented upon in these columns, makes one and possibly other larger molecular weight precursors called proinsulin. It is composed of the two chains of insulin connected to an additoinal polypeptide forming a single chain polypeptide. Because of the presence of insulin within the proinsulin molecule there is cross-reactivity within the insulin assay for proinsulin and proinsulin-like compounds. However, the biological activity of proinsulin is only 1/15th that of insulin. Measurements of proinsulin have been found to be abnormally high in patients with insulin-secreting tumors. Similarly, a large molecular weight

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glucagon compound has been extracted from pancreatic tissue and has been found circulating in the plasma of patients with tumors as well as normal volunteers ("pro-glucagon"). Similar large molecular weight precursors have been found in other endocrine tissue including that involved in growth hormone, prolactin, parathormone, renin, ACTH and thyroid stimulating hormone production.

The association of benign pancreatic islet cell tumors with various other hormone overproduction states has been linked together in the multiple endocrine adenoma syndrome. As previously reported in this column (*Arizona Medicine,* June, 1975), this syndrome involves several glands besides the pancreas. The association of pituitary and parathyroid tumors with pancreatic adenomas has been classically described in the multiple endocrine adenoma syndrome Type I.

The hypercalcemia which can be thus seen with this syndrome and that associated with islet cell carcinoma can be of multiple etiology. Obviously, excess parathormone production from an associated parathyroid adenoma or an ectopic source of parathormone can produce such a situation. For instance, Benson, et al,[4] determined the serum immunoreactive parathormone concentration in 108 unselected hypercalcemic patients with malignant disease utilizing a sensitive radioimmunoassay system that is specific for the carboxyl terminal region of the parathyroid hormone molecule. Measuring the predominant, but biologically inactive fragment of parathormone, 95% of this patient group was found to have abnormally high serum PTH concentrations for the concomitant serum calcium level. The authors commented on the heterogeneity of parathormone and suggested that their studies support the idea that ectopic hyperparathyroidism is present in the overwhelming majority of patients with hypercalcemia and another underlying tumor.

On the other hand, additional causes of hypercalcemia in patients with malignant tumors have accrued from the studies of Powell, et all.[5] These investigators have found a low incidence of parathormone or parathormone-like fragments in the circulation of patients with cancer and hypercalcemia. In vitro studies have also demonstrated that cultured tumors can synthesize a substance which has calcium mobilizing activity. Prostaglandin-like compounds have been incriminated in this phenomena and several case studies have been reported which show that the hypercalcemia in these conditions can be controlled with Indomethacin, a prostaglandin inhibitor. In addition, other compounds such as sterols and vitamin D-like substances have been shown to be produced by various kinds of malignant tumors which may then be responsible for the hypercalcemia in addition to that produced directly by tumor metastasis.

The treatment of malignant islet cell tumors, per se, has progressed somewhat in the past ten years although we are still groping for the ultimate cure. Streptozotocin has resulted in remarkable symptomatic improvement in patients with malignant islet cell tumors. In 1968 Murray-Lyon, et al,[6] demonstrated a decrease in the size of hepatic metastasis and a decreasing production of various hormones such as insulin, gastrin and glucagon. The clinical experience with Streptozotocin in 52 patients was recently analyzed by Broder and his associates. The drug was given intravenously on a weekly schedule of 0.6 to 1.0 grams per meter square body surface area. Biochemical response were seen in 64% of patients and measurable decrease in tumor size was seen in 50% of the cases. Inhibition of insulin secretion occurred two to three weeks after drug administration and a gross doubling of the median survival time was shown for the responders as compared with the nonresponders.

Additional drugs have been utilized for the oversecretion of insulin and glucagon in these syndromes and these include Dilantin, Diazoxide and Somatostatin (a recently synthesized hypothalamic pituitary hormone). These agents have not been shown longterm to produce a regression of tumor size per se, but can inhibit hormone secretion and give rise to symptomatic relief.

The treatment of hypercalcemia when it occurs with malignant or benign islet cell tumors consists initially of adequate hydration and forced solute diuresis. Depending on the cause of the hypercalcemia, the patient's general overall condition and the degree of systemic involvement determines which additional measures should be employed. For instance, the use of oral phosphates, parmacological doses of steroids. parenteral Mitramycin,[7] thyrocalcitonin and intravenous Furosemide with solute replacement[8] have all been employed in various combinations.

Recently Indomethacin has also proven helpful in selected patients.

In many patients definitive therapy by means of surgery or radiotherapy can provide a more prolonged remission.

In summary then, islet cell carcinomas are tumors which can produce clinically very diverse syndromes associated with the overt production of one or more hormones. These tumors are usually fast growing but can be compatible with a prolonged life span and are often difficult to treat because of their location. Also the specific chemotherapeutic agents that are available are limited by both their potency and their toxicity. Inhibition of hormonal production by various agents has been tried, however tumor growth is not usually prevented. Associated conditions such as hypercalcemia need to be treated although there may be more than one etiology involved in any one patient with such a tumor.

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AM *Obstetrics and Gynecology*

WHAT HAS HAPPENED TO THE CLINICIAN IN OBSTETRICS?

WILLIAM SUSONG, M.D.
DONALD J. ZIEHM, M.D., EDITOR

It appears to me that marked inconsistencies exist in the practice of obstetrics today. On the one hand we have the Back-to-Nature group of patients who consider their obstetrician an adversary to be watched very closely so that he does not trick them and use something unnatural to the birth process such as analgesics, block, amniotomy, etc. To these patients, the obstetrician is just a hurdle the patient and her husband must clear in order to have access to the delivery facilities. Many of this group would actually prefer to deliver at home if they could only find a physician willing to assume the responsibility of the home delivery and relieve them of some of the guilt in the event something goes wrong. These patients are somewhat difficult to find when you enter the labor room, for all you can see is the husband hovering over a mound of pillows. However, the breathing pattern, which is similar to the sound of a surfacing whale, will lead you to the patient's head. Then, if you are permitted to examine the patient, it requires another five minutes to unpack her for positioning. If you want to draw some stares of disgust from this patient and her mate, just ask for an amnihook.

In contrast, we have the group of patients who are managed by the laboratory-oriented physicians. The pregnancy is diagnosed with a gonadotropin assay, doptone or ultrasound. The progress of pregnancy is followed by serial ultrasound measurements, several amniocenteses, multiple blood counts, analysis of several gallons of urine for estriol and finally oxytocin challenge tests near term. This patient is difficult to see in the labor room for she has a pump connected to her IV, a large monitor strapped to her abdomen, a wire protruding from her vagina and a large piece of hardware nearby spitting out reams of graph paper with flashing lights and a beeping signal indicating the presence of the fetal heart rate. If you are fortunate, you may be permitted as the attending physician to make your way to the bedside where the nurse and residents are reviewing a change in the graph which may or may not be significant. One thing is for sure, they are not interested in your appraisal of the situation. After all, what do you

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Reprint requests to Dr. Ziehm, 1728 W. Glendale Ave., Phoenix, AZ 85021 know about late decelerations or a cord compression pattern? In this situation, the patient's husband does well to see his wife with binoculars from the corner of the room, much less be at the bedside to hold her hand.

On the one hand, we have the "Do-it-yourself movement" which is characterized by those who demand little or no participation by the obstetrician. On the other hand, we find the "Ivory tower" approach with multiple tests and use of electronic devices for almost everything. Is there no longer a need for the experienced clinician who has learned to use his eyes, ears and hands to care for his patient?

"Progress," mused Mark Twain, is composed of "all the modern inconveniences." The concept of what constitutes good obstetrical practice has changed markedly during the last ten years. The dramatic decrease in the perinatal mortality rate can be directly attributed to the development and growth of perinatal medicine as a subspecialty. Although increased fetal survival is certainly a highly desirable goal, the practice of "laboratory and electronic obstetrics" has to some degree dulled the visual and tactile senses of the modern clinician (the lament of the above author). Perhaps one day the obstetrician will be relegated to a control room, his primary function being to examine and evaluate laboratory reports and monitor tracings, leaving the more "routine" aspects of obstetrical care (prenatal visits, management of labor and delivery, etc.) to obstetrical technicians.

The ascent of the consumer has placed an additional burden on today's obstetrician. While most of us are willing to discuss our plan of management with patients, the fuse ignites when an "educated couple" promptly informs us that our way is "not natural," "dangerous" and "undesirable," contrary to the "right way" as taught by the childbirth instructor. Time honored concepts of the patient-physician relationship are being seriously challenged. Our medical knowledge and obstetric judgment suddenly seem suspect, but the medicolegal liability remains unchanged in the eyes of the consumer.

The author has scratched at several sores that are causing discomfort to many obstetricians. Although most would be unwilling to admit to these sentiments, perhaps many more are feeling similar pressures. Once learned, it is difficult to change one's attitudes and methods of practice. Indeed, many question the necessity. Each practitioner must search his own mind for the proper balance between conventional obstetric practice and the changes constantly occurring as modern technology forces modification of these traditional methods.

Editor

AM *Seminars in Chest Medicine*

BRONCHODILATOR THERAPY

ROBERT J. CLARK, M.D., F.A.C.P.

This continuing series of articles entitled "Seminars in Chest medicine" will attempt to keep the reader abreast of developments in the broad field of pulmonary diseases. The format used will be that of brief succinct reviews written by the editors as well as guest contributors. Areas of controversy as well as practical chest medicine will be explored. We hope that these reviews will be of value in promoting continuing education for certification examinations as well as a forum for new and controversial issues. The editors welcome comments and discussion from our readers.

ROBERT J. CLARK, M.D.
LYNN M. TAUSSIG, M.D.
WILLIAM C. WEESE, M.D.

The tracheobronchial tree in asthmatics is hypersensitive to many different stimuli[1] including histamine, methacholine, acetylcholine, prostaglandin $F_{2\alpha}$ some pollutants, and occasionally exercise. The milieu to which the lung is exposed is multifactorial and very complex. The pharmacologic armamentarium for the treatment of reversible airways disease has greatly expanded as has our understanding of the interrelationships of the various mediators, inhibitors, and biochemical agonists. The purpose of this brief review is to place in perspective the fundamen-

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From: 2910 N. Third St., Phoenix, AZ 85012

tals of the asthmatic response and the role of biochemistry in its pathogenesis and treatment.

The number of known mediators involved in the asthmatic response has not greatly increased but our understanding of the disease has expanded considerably. This understanding is probably primitive and in time other compounds and responses will be discovered. Histamine, serotonin, bradykinin, acetylcholine, slow reacting substance of anaphylaxis (SRS-A), prostaglandin $F_{2\alpha}$ and alpha adrenergic stimulation cause smooth muscle constriction and therefore bronchospasm.[1-4] There is an apparent imbalance of the autonomic nervous system[1, 5] and of the synthesis of prostaglandins E^2 and $F_{2\alpha}$.[6, 7] The naturally occuring agents which cause smooth muscle relaxation and therefore bronchodilation are beta adrenergic compounds such as epinephrine, corticosteroids, the prostaglandin E series and naturally occurring kinin inhibitors.[1] Pharmacologic agents used to augment the various biochemical events leading to bronchodilation are beta adrenergic stimulants, cromolyn sodium, corticosteroids, xanthines, anticholinergic agents, and prostaglandin E_2. Other agents currently being investigated are RS-7540,[8] a drug similar to that of cromolyn sodium, and synthetic polypeptides which block the attachment of immunoglobulin E (IgE) to mast cells.

Most bronchodilators depend on interaction with one of two systems in the tracheobronchial tree: bronchial smooth muscle and events before and after the release of histamine, SRS-A, and kinins from tissues. The initial events are quite different but the final common pathways, those of alteration in the intracellular levels of cyclic nucleotides, are identical.[9] In our current state of knowledge, we place the cyclic nucleotides in the center stage and, in all biochemical manipulations, attempt either to increase the intracellular concentrations of cyclic adenosine 3', 5' phosphate (c-AMP) or to decrease the levels of cyclic guanosine 3', 5' phosphate (c-GMP). Another way to view the actions leading to bronchoconstriction is in the framework of the various types of immunologic response. Type I response is an immediate histamine, IgE, or cholinergic mediated response. The type III delayed response depends on the formation of immune complexes and responds poorly to bronchodilators. It is blocked by cromolyn sodium and corticosteroids.[10]

ADRENERGIC STIMULANTS AND BLOCKERS

Epinephrine, the oldest available adrenergic stimulant, introduced in 1904, has both alpha and beta adrenergic activity. The beta activity is beta$_1$ (peripheral vasodilation and cardiac stimulation) as well as beta$_2$ (bronchodilation).[11, 12] We have witnessed the synthesis of many alpha and beta stimulants and blockers in recent decades. Isoproterenol and ephedrine, which are widely used, have no alpha activity but have both beta$_1$ beta$_2$ activity. We have recently seen the introduction of agents with a great selectivity for beta^2 receptor sites.[12] The first commercially available was isoetharine with a beta^2:beta$_1$ activity ratio of approximately 2:1. Metaproterenol has a beta$_2$:beta$_1$ ratio of 5:1. Terbutaline, salbutamol, fenoterol, and other agents are currently being evaluated. Terbutaline is presently available in oral and subcutaneously injectable forms. These compounds are more highly selective for beta$_2$ receptor sites with the beta$_2$:beta$_1$ ratio being approximately 200:1. These act primarily by increasing intracellular levels of cyclic AMP and lead to bronchodilation. Alpha stimulation in the lung, however, leads to elevation of cyclic GMP levels and antagonizes the effects of the cyclic AMP, thereby causing bronchoconstriction.[1, 11, 13]

Blocking agents for the alpha and beta receptor sites have been synthesized. Phentolamine, dibenzylene, and thorazine block alpha receptor sites and inhibit the stimulatory effects of alpha agonists by inhibiting the increase of cyclic GMP. Propanolol blocks with beta$_1$ and beta$_2$ receptor sites whereas practolol (not available in this country) blocks selectively only beta$_1$ sites.[14, 15] Propanolol should be used in asthmatic patients with great caution because of the precarious balance existing between the various mediators. Propanolol induced bronchospasm in the asthmatic can be severe and relatively resistant to conventional therapy. Atropine is of value in the treatment of propanolol induced bronchospasm.[16] *Beta$_2$* stimulation also improves mucociliary transport, which may be of clinical importance.[17] These agents may be administered by a variety of routes, depending on their absorption and toxicity.

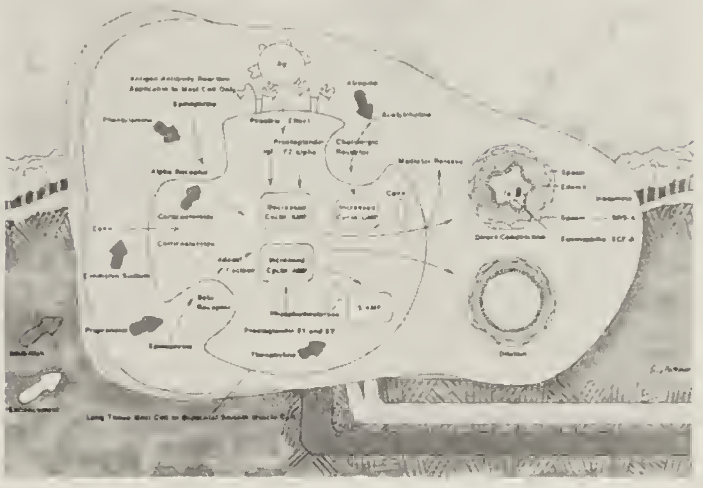
XANTHINE COMPOUNDS

Naturally occurring alkaloids of the xanthine class have been used for many years in the treatment of asthma. The oldest, most widely used of these is theophylline which is a potent bronchodilator. Because of the insolubility of theophylline, several salts and derivatives such as aminophylline (theophylline ethylenediamine) dyphylline, and choline theophyllinate have been synthesized. Their mechanisms of action, however, remain identical to that of theophylline. Theophylline is a potent inhibitor of the phosphodiesterase enzyme responsible for the degradation of cyclic AMP. The inhibition of this enzyme leads to increases in the intracellular levels of cyclic AMP and then to bronchodilation. It has been suggested that there might be either an additive or perhaps synergistic effect with concurrent use of beta adrenergic stimulants. One recent study[18] seems to refute this claim, but the dosage of both xanthine and beta stimulant (terbutaline) was large and perhaps the effect was minimized.

ANTICHOLINERGIC AGENTS

Stimulation of irritant receptors in the airway can cause reflex bronchoconstriction in man.[19, 20] Asthmatic patients have hyperirritable airways and respond to much less stimuli than do normal individuals.[21, 22] Reflex bronchoconstriction is greatly inhibited by atropine sulphate.[23] Histamine is liberated from lung tissue on a reflex basis and on exposure to specific antigens.[24, 25] Atropine has for many years been used in the treatment of asthma. Inhaled atropine and similar alkaloids have been used in Asia and India in the form of strammonium (Jimson weed) cigarettes and powders.[26] With the introduction of epinephrine in 1904 and the development of problems with the dosage of atropine, the use of this form of therapy was sharply curtailed.[1, 11, 26, 27] A resurgence of interest in anticholinergic drugs in the treatment of asthma has become apparent.[11, 13, 28] The theoretical disadvantage of inspissation of secretions seems not to be a clinical problem,[29,30] but long term controlled studies are necessary to evaluate this possibility further.

Acetycholine causes an increase in cyclic GMP and the release of bronchoconstricting mediators, such as histamine.[1,5] Conversely, the blockage of acetylcholine with atropine or other anticholinergic compounds will inhibit this increase in cyclic GMP and its attendant bronchoconstriction.[1, 5, 9, 11, 28] Precisely what future role atropine will play in the treatment of asthma remains to be determined.



PROSTAGLANDINS

Since the discovery of prostaglandins, much data on their use have been collected. The prostaglandin E (PGE) and F (PGF) series have been extensively studied. The lung is not only a target organ for these compounds but plays a major role in their metabolism.[30, 31] The ratio of PGE:PGF in the normal lung is fairly constant but in the anaphylactic animal lung, much greater amounts of PGF are produced.[7] The asthmatic patient requires 10,000 times less inhaled $PFG_{2\alpha}$ to cause bronchoconstriction than the normal person and is also more responsive to bronchodilation by PGE_2.[6] Bronchospasm in healthy subjects and asthmatic patients has been reported in studies involving the intrauterine installation of $PGF_{2\alpha}$ when used for therapeutic abortion.[32] Clinical trials of PGE_2 are being planned for the near future.

Prostaglandins also exert their effects on the cyclic AMP system. The PGF series causes a decrease and conversely the PGE series an increase in the intracellular levels of cyclic AMP.[33] The exact mechanisms of these phenomena are unknown. The use of PGF compounds in allergic or asthmatic individuals should be undertaken with extreme caution.

CORTICOSTEROIDS

Corticosteroids have been used in a multitude of clinical disorders including asthma. To this date, their mechanism of action is largely unknown. They do not, however, block the immediate or type I immune mediated histamine response. They have a latent period of action of from four to six hours, and they alter the delayed type III immune response.[1, 10, 11] Many asthmatics demonstrate a bimodal response to allergens[1, 10] with both an early type I and a latent type III response. Corticosteroids often are required by adult asthmatics for maintenance of a normal existence but to date their use has been clouded by the too well-known unwanted side effects. The use of sparingly soluble aerosolized steroids has largely eliminated the severe major side effects.[34, 35] The sparingly soluble aerosolized steroids are beclomethasone and triamcinolone acetonide. Some effects of these drugs are to increase the concentrations of intracellular cyclic AMP, stabilize lysosomes,[1, 11] enhance beta stimulation,[38] inhibit alpha stimulation by direct alpha blockade,[1, 10, 11, 13] and stabilize membranes.[1, 11] There are no data that suggest that they inhibit the release of mediators from mast cells.[1]

CROMOLYN SODIUM

The introduction of cromolyn sodium (DSG) was a great advancement in asthma therapy. It is a well tolerated, relatively nontoxic, and potent drug. It inhibits both the early type I and late type III immune response.[1, 10] It also blocks the occurrence of exercised induced asthma.[1, 10, 11] The mechanisms of action proposed include the inactivation of sensitizing antibody[1], desensitization of mast cells by inactivation of mast cell sensitizing antibody through combination with the first antigen encountered[36, 37] and interaction with calcium at the membrane level.[36] The incidence of serious side effects of this drug is very low and usually clinically insignificant. Disseminated vasculitis has been reported as a serious side effect. The oral form of DSG is being studied along with other analogs with similar modes of action.[8]

The physician today has a much greater effective array of drugs with which to treat the asthmatic patient. In the years to come, we will probably see newer, even more effective and less toxic drugs introduced.

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Radiology
Case of the Month

RADIOLOGY CASE NO. 12

J. C. BJELLAND, M.D.

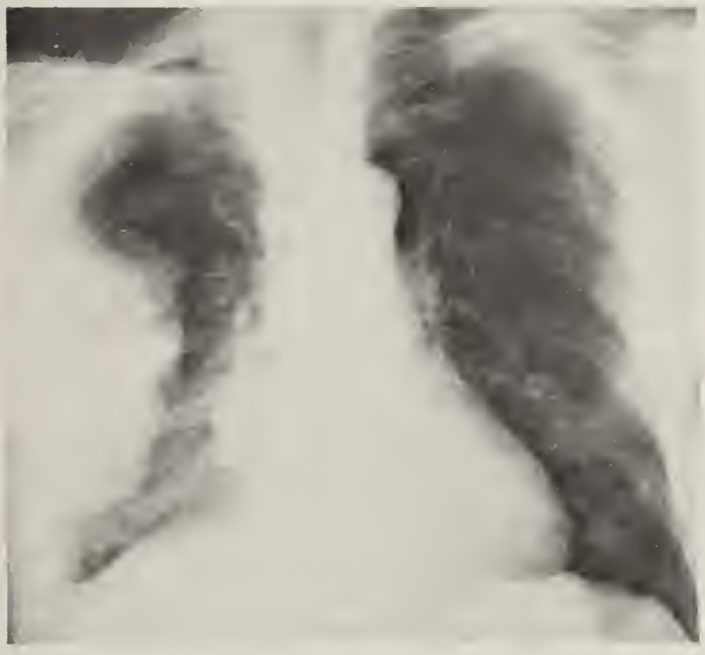

FIGURE 1
The right hilar adenopathy is an incidental finding, which can be diagnostically disregarded.

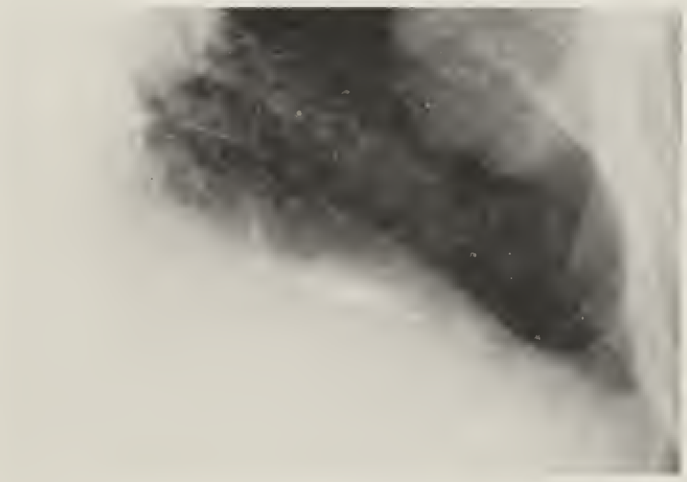

FIGURE 2
Coned down view of the right lower lobe.

A 59 year old male presented in the emergency room with complaints of cough and dyspnea on exertion. The patient denied having any significant past medical history, but did state that his occupational history for the past twenty-five years had been that of a miner. Physical examination revealed dullness to percussion in the right axillary line. Based primarily on the radiological appearance of the chest above, what is the best diagnosis?

From: Dept. of Radiology, U of A, College of Medicine, Tucson, AZ 85724

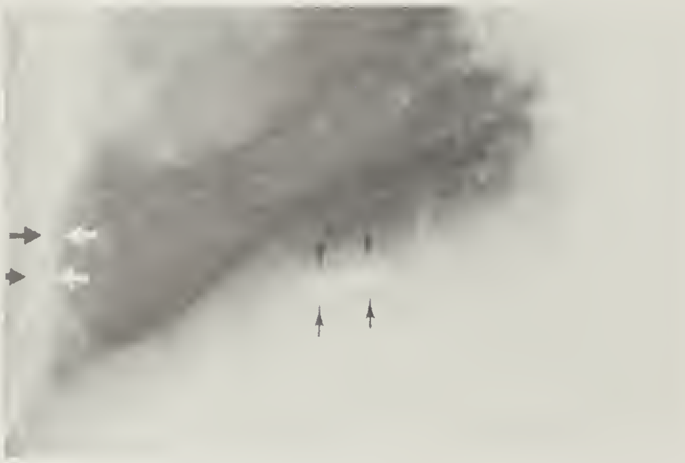

FIGURE 3
Asbestosis and Right Pleural Mesothelioma.

The radiographic findings are: 1.) a large, broad, pleural-based, irregular lobulated mass extending from the right pleural line; 2.) pleural thickening in the right lateral costophrenic angle (see opposing paired horizontal arrows in the photograph above); 3.) the presence of a right diaphragmatic curvilinear pleural calcification (see opposing paired vertical arrows in the photograph above); and 4.) right lower lobe interstitial fibrosis.

The presence of pleural thickening, pleural calcification (particularly along the diaphragmatic surface), and interstitial parenchymal disease with predominance in the lower lobe are the radiologic hallmarks of asbestosis. In light of these findings, the associated mass lesion fairly confidently may be called a mesothelioma, due to an increased incidence in association with asbestosis. The patient does have an occupational history of being an asbestos miner in Quebec, Canada, and the mass lesion was proven on needle biopsy to be a mesothelioma with asbestos bodies present within it.

Asbestosis is the most important member of the pneumoconioses known as the silicatoses, in which salts of silicic acid are the exciting agents. Asbestos is a relatively indestructable mineral, whose fibers are ribbon-like and measure approximately 3u X 100u when airborne. The unique characteristic of the fiber is that it produces pleuro-pulmonary disease which has a distinct propensity for an associated subsequent development of neoplasia. The development of pleuro-pulmonary disease in asbestosis depends on a combination of the degree and length of exposure to its fibers. Usually the disease becomes manifested after lying in a state of latency for 20 to 40 years after the initial exposure.

The pulmonary parenchymal component of the disease is confined primarily to the lower lobes and consists of an interstitial fibrotic reaction that develops secondary to the liberation of silicic acid by the decomposing asbestos fibers. The fibrosis is chiefly located in the bronchiolar wall and involves the alveolar cells, leading to a physiologic alveolar-capillary block. Consequently, the primary functional abnormality is inadequate gas exchange.

The pleural component of the disease is thought to be due to penetration of the visceral pleura by the asbestos fiber, with irritation of the parietal pleura ultimately resulting in hyaline plaque formation. Pleural biopsy will establish the diagnosis by demonstrating the presence of asbestos fibers in the pleural plaques. Freundlich and Greening noted in 56 cases of proven asbestosis that pleural manifesations of the disease dominate the radiographic picture. They found 48% of their asbestosis patients to have pleural plaques or thickening only, while 41% of those patients had combined pleural and interstitial parenchymal disease. Only 11% of their patient group had parenchymal changes alone. Pleural calcifications were observed in 21.4% of their 56 cases. However, in patients with over forty years exposure, the prevalence of calcified pleural plaques is greater than 50%. Both calcified and non-calcified pleural plaques were characteristically found to occur over the antero-lateral chest wall inferiorly and over the central diaphragmatic domes.

As exemplified by this case, of all non-neoplastic pulmonary disease, asbestosis undoubtedly has the highest incidence of associated neoplasia of all types, especially bronchogenic carcinoma and mesothelioma (both pleural and peritoneal). The average risk of a twenty year asbestos worker developing lung cancer is ten times that of the general population. If that same twenty year employee is also a smoker, a carcinogenic synergy is noted, so that then the risk for that patient developing cancer is ninety times that of the general population. Unfortunately, in agreement with this data is the fact that the typical asbestos related trades (e.g., pipe fitting and shipyard industry, brake lining manufacturing, etc.) show neoplasia rates similar to that of the asbestos miner.

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Renal Disease

ANTIBIOTICS AND THE KIDNEY — II

Antibiotic Induced Nephrotoxicity

GEORGE M. NICKAS, M.D.

A wide variety of antibiotics produce renal damage due either to direct nephrotoxicity or to hypersensitivity reactions. The frequency and potential of renal damage depends upon a number of factors. These include the chemical structure, the dosage used, the simultaneous use of other nephrotoxic agents, the pre-existence of renal disease. Nephrotoxicity due to antibiotic therapy is largely reversible upon discontinuation of the agent. However residual damage may be permanent, e.g., amphotericin B. The clinical and pathologic characteristics of antibiotic nephrotoxicity varies depending upon the drug or drug group in question. Finally, certain antibiotics with nephrotoxic potential also require dosage reduction in the presence of pre-existing renal impairment to avoid potentiating nephrotoxicity as well as extra renal toxicity. Examples would include the aminoglycoside antibiotics. These latter groups of antibiotics require particular care and frequent monitoring of renal function during therapy. Schreiner's review on toxic nephropathies summarizes earlier experience with antibiotic-induced renal damage. Since then the introduction of newer groups of antibiotics such as the cephalosporins and further experience with the older groups requires some review. This review deals selectively with certain antibiotic groups.

The following antibiotics have not been shown to be nephrotoxic:

chloramphenicol, erythromycin, lincomycin and clindamycin.

The major antibiotic groups with nephrotoxic potential are discussed below.

Certain penicillins (penicillin-G, methicillin, and ampicillin) may cause an acute allergic interstitial nephritis. This represents an immunologically induced disorder and probably not strictly a nephrotoxic effect. This syndrome generally occurs with use of massive doses of penicillin-G, methicillin, or ampicillin and generally is reversible upon discontinuation of the agent. The syndrome includes oliguric or non-oliguric renal failure with fever, rash, eosinophilia and urinary abnormalities including hematuria, proteinuria, and eosinophilinuria, Pathologically, acute interstitial nephropathy is seen and anti-tubular basement mebrane antibodies have been demonstrated with nephritis due to penicillin-G and methicillin.

The cephalosporin antibiotics vary considerably in nephrotoxic potential. Cephalothin, cephalexin, and cephaloridine all may cause renal damage. The pathogenesis of cephalothin toxicity is not clear; the incidence of toxicity would appear to be quite low in view of the scarcity of reported incidents. The most common clinical picture is that of oliguric or non-oliguric acute tubular necrosis. However a second syndrome of renal failure associated with rash, history of previous penicillin allergy, eosinophilia and histologic evidence of interstitial nephropathy have also been described. This latter picture strongly suggests a hypersensitivity nephritis similar to that described above with the penicillins. In addition cephalothin-gentamicin combination nephrotixicity is recognized and usually leads to more severe acute tubular necrosis.

Reported cephalexin nephrotoxicity is rare and characterized by a syndrome similar to that of cephalothin, with previous penicillin allergy, non-oliguric renal failure with hematuria and proteinuria, rash and fever, again suggesting a hypersensitivity reaction. Renal histologic study is not available. Cephaloridine is the most renal toxic cephalosporin available to date, causing acute tubular necrosis. Its toxicity is dose related and doses not exceeding four grams per 24 hours are recommended in patients with normal renal function. Reduced doses are recommended with pre-existing renal impairment as well as avoidance of simultaneous nephrotoxic agents.

The polymixin antibiotics (polymixin B and colistin) are recognized nephrotoxins producing renal insufficiency, oliguric or non-oliguric acute tubular necrosis. Adverse renal effects have been reported in as many as 20% of utilizations with colistin (sodium colistimethate) in one large series. In addition, peripheral neuropathy and myoneural dysfunction with respiratory paralysis are

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potentially serious complications. Finally both agents require major reduction in dose with pre-existing renal impairment thus requiring particular care and frequent montioring of renal function during usage.

Aminoglycoside antibiotics have, in common, potential for renal and ototoxicity. Gentamicin renal damage occurs in approximately 0.5 to 2% of utilizations, generally mild, and invariably reversible. Azotemia, cylinduria, proteinuria are the usual manifestations. In the more severe cases acute tubular necrosis with or without oliguria may occur. A recent association of cephalosporin and gentamicin combination causing anuric tubular necrosis has been noted and suggests potentiation of nephrotoxic effects between these two agents. Tobramycin similarly is nephrotoxic and ototoxic and incidence of adverse effects and the safety compared to gentamicin usage requires further experience with this agent. The possible potentiation of adverse effects with cephalosporin await further experience as well. However, quite clearly, the use of gentamicin and tobramycin as well as other aminoglycoside antibiotics such as kanamycin and neomycin require monitoring of 8th cranial nerve function and renal function for prompt recognition of toxic effects.

The tetracylcine group of antibiotics produce at least three recognized types of renal disorder. Probably the most common is the further rise of BUN in patients with pre-existing azotemia. This seems to be somewhat more frequent with the simultaneous use of diuretic therapy. There may be associated metabolic acidosis as well as an overt uremic syndrome. This had generally been attributed to the anti-anabolic effect of tetracycline on protein synthesis; however a number of patients are observed to have a rise in serum creatinine as well, implicating a direct nephrotoxic effect. This rise in BUN and creatinine may also occur in patients with pre-existing normal renal function. The second major syndrome is the Fanconi syndrome caused by out-dated tetracycline characterized by glycosuria, aminoaciduria, metabolic acidosis, hypokalemia and azotemia. This syndrome is reversible with discontinuation of the tetracycline although mild histologic and functional changes may persist for months afterwards.

The third renal disorder has been described with demeelocycline (Declomycin) nephrogenic diabetes insipidus which is dose dependent and reversible. The polyuria and loss of concentrating ability may persist for some period of time following discontinuation of demeclomycline, however. The cause of this syndrome appears to be an antibiotic-induced defect in renal tubular response to anti-diuretic hormone.

Mention must be made of the long recognized nephrotoxicity associated with amphotericin B therapy. Early renal dysfunction is characterized by appearance of renal tubular acidosis with defect in renal concentrating ability. In addition rise in BUN and creatinine commonly occur. Hypokalemia is also a frequent feature. A variety of histologic changes are noted including nephrocalcinosis although some studies would indicate no consistent relation between the severity of histologic change and the total dose of amphotericin B administered. With larger doses renal impairment may be irreversible and total renal failure can occur with the massive doses. The use of mannitol has been suggested with amphotericin B in order to reduce nephrotoxicity. The use of combination therapy in reducing renal toxicity has given conflicting results. The simultaneous use of mannitol however does not result in increased excretion of amphotericin and no differences in blood level determinations are evident in the amphotericin vs. the amphotericin-mannitol treated groups. Further experience on the possible protective effect of mannitol would seem desirable.

SUMMARY:

A variety of antibiotics can produce adverse renal effects. Fortunately the vast majority of these effects are reversible with discontinuation of the antibiotic in question. This survey has reviewed highlights of antibiotic-induced renal damage with antibiotics both new and old.

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Department of Health Services

INFLUENZA IMMUNIZATION CAMPAIGN

SUZANNE DANDOY, M.D.
DIRECTOR

Periodically, events occur in public health that excite the imagination and challenge the ingenuity of those responsible for program development. One such event occurred earlier this year when "swine flu" was diagnosed among the recruits at Fort Dix, New Jersey. From this episode has arisen one of the most controversial and far reaching programs in preventive medicine since the polio campaigns in the late 50's and early 60's. Our immunization delivery system is being challenged as never before.

Plans call for the voluntary immunization of approximately 90% of the target population before the end of November. The target population includes everyone except those allergic to eggs and some pediatric populations which have not yet been defined.

Development of an immune population through administration of vaccines to large segments of the population has been attempted against polio, measles and rubella in recent years. Such mass campaigns have demonstrated that diseases can be controlled effectively by the use of vaccines that induce long lasting immunity. Influenza does not produce a long lasting immunity, however. The situation is further complicated by the fact that influenza viruses undergo variations, necessitating continual changes in the vaccines. And, about every ten years a major change in the virus occurs, signaling widespread epidemics and increased mortality, especially among high-risk groups.

In February of this year an outbreak of influenza occurred among recruits at Fort Dix, New Jersey. Epidemiological studies revealed there were at least two viruses active: A/Victoria and A/New Jersey. Although only 12 cases of influenza were confirmed, extensive blood testing indicated that probably several hundred recruits were infected during the outbreak. No spread into the surrounding community was demonstrated.

The A/New Jersey strain is identical to the virus that causes influenza in swine. Swine influenza has rarely occurred in people, at least in the past 50 years. Person-to-person transmission of the swine virus was a departure from the usual pattern and raised the possibility of a "seeding" of the virus into the human population. The serological characteristics of the virus were found to be the same as those of the virus to which the 1918-19 pandemic has been attributed. Consideration of these factors led to the recommendation to conduct a massive immunization campaign.

Between mid-July and the end of November two influenza vaccines will be available. A bivalent containing A/Victoria and A/New Jersey strains will be produced for use among high-risk groups. A monovalent vaccine containing A/New Jersey will be available to immunize the general population against "swine flu." Neither of these vaccines will be marketed through the usual channels and practicing physicians will be unable to obtain the vaccines from their usual sources. Every dose manufactured will be purchased by the federal government and distributed through the state health departments. In Arizona the vaccine will be distributed by the Department of Health Services to the County Health Departments.

Several vaccines studies are underway to ascertain reactions and antibody response to the vaccines. Also to be resolved are the matters of immunization of pediatric groups and the risk responsibility when the vaccines are administered. Answers to these and other questions will be forthcoming in advance of the initiation of the program.

This program is a tremendous undertaking. However, it does present the opportunity to test a new concept in communicable disease control: that an immediate all-out effort can blunt the effect of a virus with the potential for producing a national epidemic, and perhaps even a pandemic.

Practicing physicians can make significant contributions to the success of the program by working closely with their county health departments who will bear the major burden of administering the vaccines.

Editorial

NARCISSISM

WILLIAM B. McGRATH, M.D.

All of the personality disorders and most psychiatric illnesses have one trait in common.* It is narcissism.

We do not need a battery of psychological tests to identify this trait. We do need to observe how constant and how appallingly widespread it has become.

You might make a statement, any statement; such as, "We are buying a new house." Notice the responses.

"Is that right? Well, we've been looking at condominiums."

"My house needs a new roof."

"I wish I could afford a place of my own."

An essay was submitted for publication. In two pages the words "I", "me", "my" or "mine" appeared fifty-three times! The rejected writer will be defensive, rationalizing, "Is it wrong for me to express myself?"

At a gathering a man said, "We have just found out that my wife has leukemia." Again, listen to the comments:

"I'm sorry to hear about it. I had an uncle who died from it."

"Is that a fact? I thought I had it once but it turned out to be mononucleosis."

"That's too bad. Say, do you know anybody who might be in the market for a good quarter horse?"

Some people said and will say nothing at all. They will behave — and expect him to behave — as if nothing is wrong and nobody cares.

Sociologists write about alienation. Can you understand why the man feels disconsolate and alone and even a bit misanthropic?

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*Just their listing would support the proposition, if space permitted.

In some ways egocentricity is being cultivated. Parents are expected to show interest in their children's activities and moods. Little or no attention is paid to teaching the reverse, the reciprocal. In a classroom some of the boys and girls seem to spend most of the time preening themselves, combing their long hair. Or witness the insufferable conceit of the advertising model on television: "I like me because I use such and such." Courses in salesmanship stress that you have to sell yourself. In psychodrama and in group therapy, the individual is encouraged to tell others all about himself.

Narcissism is an insidious parasite in politics. The office-seeker or office-holder "projects an image," plays constantly to the audience. If he is posturing most of the time then he cannot think or go about his work with the natural seriousness it should demand.

The individual who is most self-centered is paradoxically the least aware of it. And we are hesitant and too awkward to point it out to him. *It is a wounding fallacy to suppose that one is sharing another person's triumph or sorrow by mentioning one's own.* We protest that acquaintances have to know more about us if they are going to like us or become friends. We all make this mistake, turning the topic back to ourselves, and it is a habit hard to break.

Rare and to be cherished is the person who can pay a compliment or express pure sympathy or convey genuine interest in someone other than himself. This is an area in which the professions can at least provide a helpful example. The reporter wants to know how something happened and to whom, and he at least has to get the correct spelling of the names. The clergyman cares — and knows how very important it is to say so. The lawyer and the doctor learn to ask questions and to listen attentively to answers and to express concern. Would that we were as considerate in our private lives!

There was wisdom as well as sweetness in the old practice of kneeling at bedtime and simply praying that others might be blessed. This was the opposite of narcissism.

We have written of the need of a hearing. Especially during crises in their lives, people do derive consolation and strength from the fact that someone listens with real concern and remembers to express it. This is worth more than psychoanalysis and all the pills in the pharmacy.

PREDICTIVE DISABILITY EVALUATION

WALTER V. EDWARDS, M.D.

Comprehensive medical care is certainly not a new concept. Patient's with catastrophic medical illnesses and injuries are seldom deprived of such care. However, one group of patients is rarely offered such care until chronic disability has been almost irreversibly established by the passage of time, vegetation of the patient and medical focus on local pathology, following a relatively trivial injury. We refer to the "industrial low back injury."

Admittedly, most such injuries must be correctly diagnosed and treated, and most patients with lumbosacral strains, for example, respond to therapy and return to productive labor with minimal loss of time from work, a healthy outlook and little if any economic stress.

But each year, there are several hundred patients in Arizona who experience misery, pain, deprivation and confusion because of their own inadequacies combined with a failure of coordinated management of these problems by the medical profession, social services and the insurance industry, which segments of society not infrequently work at odds with each other to the detriment of the patient and with added cost of consumer goods and services through the loss experience of employers and greater cost of government services.

Because the physician is the principal contact for the patient with a medical problem for several months or years, the physician must assume a more positive role in bringing together the other resources when the doctor-patient relationships are not productive of therapeutic response, or permanent impairment and disability is predictable.

Failure of therapeutic response by the patient with a lumbosacral strain within a few days or at most a few weeks suggests a need to explore other possible reasons in the psychosocial areas, concurrent or even before exhaustive physical studies are performed, particularly when objective physical findings on an adequate physical examination are minimal or absent. Consideration of age and occupation, education and vocational experience, and exploration of interpersonal relationships at home and at work can frequently be much more productive from a

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From: 800 N. 1st Ave., Phoenix, AZ 85003

diagnostic point of view than electromyography and myelography. This requires the investment of somewhat more of the physician's time, but may save considerable time later on, and may be otherwise much less costly for all concerned. Such exploration does not require the services of a psychiatrist. The thoughtful clinician can make the process therapeutic while eliciting more information than a computerized MMPI. A potential vicious cycle may be nipped in the bud by appropriate counseling.

At the other extreme, the patient who presents early with signs and symptoms of a protruded disc, who does not respond to conservative management and who will, when stationary, probably have a degree of permanent impairment (with or without laminectomy) is entitled to evaluation and care beyond that directed to the local pathology. Consideration of age, occupation, skills (or lack of them) and the patient's role in the family and other health problems may suggest earlier attention to the need for vocational and other rehabilitation services in addition to or even instead of surgical management of the problem.

Statistically, the results of low back surgery in the industrial back case are abysmal with respect to the prevention of permanent disability. Failure (or success) is predictable early after injury through a brief assessment of the whole man and his internal and external environment. With consideration of factors peripheral to the low back in the treatment process, the physicians of our community can achieve a much higher salvage rate.

Topics Of Current Medical Interest

VD CONTROL

COMPILED BY J. C. PINTO, M.D.

1. It is almost unbelievable that a world wide runaway GC epidemic has occurred during this period when we have at our disposal diagnostic and therapeutic remedies of effectiveness never known before.[1, 2]

2. And, equally remarkable is the fact that even though the **gonococcus** has the most fastidious growth and survival requirements, it is the **number one infectious agent in this country with someone else contracting GC every 15 seconds** around the clock![2] (The **gonococcus** needs an alkaline medium not less than pH 7.2 nor more than pH 7.6; needs a temperature of 35 degrees C to 36 degrees C and is unable to survive a temperature change of 3 degrees; can only attack columnar and transitional epithelium; must have moisture and is so sensitive to drying it can only survive 1 to 2 hours on exposure to air.)

3. The gonococcus invades transitional and columnar epithelium by penetrating the spaces between the cells with subsequent involvement of the subepithelial layers. The urethra responds to this invasion with erythema, edema and finally purulent exudate.

4. The destructive aspects of GC are presumably due to a lipopolysaccharide endotoxin which is heat resistant and moderately toxic. It has been identified as a **lipopolysaccharide phosphoric acid ester.**[3]

5. Four forms of GC are recognized. **Type I** — usually found in acute GC; **Type II** — usually found in chronic GC and old laboratory cultures; **Types III** and **IV** — found in laboratories from repeated subcultures of **Types I** and **II. Only Types I and II are virulent.**[4]

6. Gonorrheal arthritis occurs as a complication in 0.1% to 0.3% of untreated patients and occurs mainly in females at a rate of 5 to 1.[5]

7. Three percent of patients who contract gonorrhea also contract syphilis. **This one fact alone is reason enough to us aqueous procaine penicillin G as the drug of choice because 4.8 mu of APPG with one gram of probenecid will abort an incubating syphilis.**[6, 7]

8. Reiter's Disease, a triad syndrome consisting of (a) non-gonococcal urogenital infection (urethritis, prostatis or cystitis. (b) non-suppurative arthritis (usually polyarticular) and (c) conjuctivities or iritis, and reported to occur in 3% of non-gonococcal urethritis cases, has not been seen at this VD Clinic. Perhaps the urethral exudate fades into insignificance with the pain of arthritis and/or ocular infection prompting such

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From: Maricopa County Health Dept., 1825 E. Roosevelt, Phoenix, AZ 85012

patients to go to their private physicians or general treatment clinics rather than a VD Clinic.

9. Although GC in the male is a relatively common disorder **"Not every urethra that drips is gonorrhea"**; our statistics reveal that only 50.74% smear or culture out GC; the remaining 49.26% treated as non-gonococcal urethritis (NGU). In 1973, England and Wales reported that the number of NGU cases exceeded the number of GC cases.[1]

10. **Non-gonococcal urethritis (NGU) is a diagnosis of exclusion—the gram stains and/or cultures are negative.** The organisms involved in NGU are: **chlymydia, T-strain myocoplasma, hemophilus vaginalis, tric agents** (Trachoma-Inclusion Conjunctivitis), **diptheoroids and mimae polymorpha varidans** — all will respond to tetracycline **but not to penicillin.**

11. There is no typical male GC or NGU urethral exudate. Dependence on clinical judgment alone can err by 50% either way. Failure to confirm by doing a simple gram stain may be some of the reason for **penicillin** failures. **NGU's just do not respond to penicillin.**

12. The real danger in not confirming by smears or cultures lies in **mis-diagnosing a GC as an NGU** thereby allowing a "female-positive-contact" to go on infecting other males — or vice versa if cultures are not performed on females.

13. Every positive male or female should be advised and afforded the opportunity to advise their contacts that they may have been exposed to VD — notification best done through an experienced CDI (communicable disease interviewer).

14. A corollary could be **"no treatment without diagnosis"** — prophylactic treatment without epidemiological follow up carries with it the **grave risk of infective contacts remaining undiscovered.**[8]

15. While it has been suggested that the incidence of **penicillin-resistant-gonococcus** increases from east to west we have not had this problem. We have had but a 1% failure rate. Perhaps the hypothesis that "maximally effective antibiotic" schedules curtail the appearance of resistant organisms,[6, 17] has worked well here. We have been on 4.8 MU APPG with 1 gram of Probenecid since January 1973.

16. The gram-negative diplococci-oxidase-positive-organisms that could be mistaken for GC unless sugars are performed are:

	G (-) Diploco	Oxidase Positive	Glucose	Maltose	Sucrose	Lactose
N Gonorrhea	+	+	+	-	-	-
N. Catarrhalis						
N. Flanescens	+	+	-	-	-	-
Mimae Polymorpha Var. Oxidans						
N. Meningitides						
N. Flava	+	+	+	+	-	-
N Subflava						
N. Sicca						
N Hemolysan	+	+	+	+	+	-
Diplococcal Rods Mimae Polymorphan-coccal	+	-	-	-	-	-
Herella Vaginicolo	+	-	- w+	-	- +	-

17. Some short gram-negative rods that may also produce a positive-oxidase reaction, may upon a very casual or too rapid a microscopic examination, appear to be diplococci — these **"diplo-bacilli"** are less likely to be confused with **diplococci** if one recalls that in the **diplococci the long axes are parallel** where as in the diplobacilli the long axes are end to end.

18. A gram-negative diplococcic stain from a positive oxidase culture grown on a Thayer-Martin (TM) plate is not prima facie evidence of GC. The following clearly indicates a need for caution; of 39 positive TM cultures, oxidase positive screened from 1,850 females in a military population, **only 8 proved to be GC when confirmed by sugar fermentation. The remaining 31 were identified** as **mimae polymorpha var, oxidans.**[9]

19. The vancomycin, colistimethate and nystatin in the Thayer-Martin media formula is supposed to reduce overgrowth of contaminating organisms and in particular is supposed to control the growth of **mimae polymorpha** variants and other **diplococci** that could be mistaken for GC. Trimethoprim lactate has also been added to control proteus and increase yields in anal cultures.

20. In a **VD Clinic** setting, an endocervical planting on TM plates which yields gram-negative diplococci-oxidase positive colonies is good presumptive evidence of N. Gonorrhea and confirmation by sugar fermentation studies should not be required as a matter of routine. Of 48 consecutive positive endocervical cultures submitted here for sugar fermentation studies, 39 were confirmed GC and 9 reported "unable to initiate growth."

21. However, in a **private setting, to avoid marital socio-legal problems, it may be advisable to treat as GC but order sugar fermentation stu-**

dies prior to making the diagnosis! The patients should be advised of the differential diagnostic problem but treated prophylactically (particularly if on or about menses) while awaiting confirmation. Sugars are routinely ordered from this clinic whenever it appears a problem may arise.

22. The onset of **gonococcal salpingitis** occurs during or immediately post mensal in at least 75% of cases. The cervix, the urethral, Skene's and Bartholin glands are the areas principally attacked in a primary infection. The infection tends to remain at the level of the endocervix until menses where if conditions are favorable, it ascends into the endometruim, spreads to the tubes, ovaries and possibly peritineum.[5]

23. For GC screening in the female, the cervical site is the single best site to obtain specimens for culture with an 85% to 90% positivity yield from infected women. Add a rectal culture at the same time and positivity rate may be increased to 98%.

24. A survey in 1968 revealed that 89% of patients treated for VD by private MD's were not statistically reported to health officials and that positive cervical cultures ranged from a low of 2.0% in private practice to 30% in VD clinics – **the same appears to be true today.**

25. Oregon State Health Department clearly illustrated the need for uniform guide lines in the treatment of uncomplicated acute gonorrhea by conducting a survey and learning that 103 practicing MD's were using some 61 different drug regimens with many inappropriate and/or suboptimal.[10]

26. **Recommended treatment for uncomplicated gonorrhea, male or female is: 4.8 MU aqueous procaine penicillin G (APPG) with one gram of probenecid – alternate** (penicillin allergies) **– spectinomycin two grams.** Long acting forms of penicillin such as benzathine penicillin G effective in syphilo-therapy have no place in the treatment of gonorrhea—they just do not achieve the high blood levels needed for a cure.

27. An essential requirement for the bacteriocidal action of APPG is that bacteria be actively multiplying. The APPG works by inhibiting the production of mucopeptides which are vital for the development of the gonococcal cell wall. **APPG just does not work well on slow growing, dormant or non-multiplying cells.**[11]

28. **The administration of a bacteriostatic drug concomitantly with APPG does not represent rational therapy since incomplete suppression of bacterial cell growth by bacteriostatic agents may inhibit the bacteriocidal action of penicillin.**[11, 12]

29. Epidemiologically, gonorrhea is an uncontrollable disease for 2 very important reasons; **first** – 80-85% of infected women are asymptomatic and **second** – vigorous control measures are practically impossible to establish and maintain because (a) of our democratic process and (b) complex human and cultural factors. **China has beat the problem – they just make an offer to their people that can't be refused!!**

30. There is one thing sure about sex education, it does nothing for the VD rate – what with the 3 P's, Pill, Permissiveness and Promiscuity. It has been argued that VD is fundamentally a behavioral disease and should be approached accordingly.

31. **The maximum recommended dose of procaine is one gram.[13] There are 120 MGMS of procaine in each 300,000 units of APPG – or a patient receives a total of 1.92 grams of procaine with each 4.8 MU of APPG.[14]**

32. Do not assume that the patient, male or female is cured just because you administered the recommended dose and/or the symptoms have subsided – **always follow up with a test of cure (TOC) and in the female always include an anal culture.**

33. A female has a 50-50 chance of contracting GC when sexually exposed to an infected partner. Therefore with any good history of exposure or reported as a contact, treat epidemiologically. That is, treat patient – do culture – if culture positive do epidemiological follow up including a **test of cure.** Our statistics reveal that out of 2,293 females stating that they were either in contact with GC or were reported as a contact to GC, 50.9% were positive.

34. Prostatic cultures are positive for GC only when urethral cultures are positive – suggesting that prostatic gonorrhea coexists only with anterior urethral infection and does not exist alone.[15]

35. With minimal screening and vigorous use of the motto "if in doubt use **spectinomycin**" we have enjoyed an extremely low penicillin anaphylactic rate. Out of 5,555 APPG treatments, only 11 known mild anaphylactic cases have occurred – a rate of only 0.2% – which is in the same ball park of national figures of 0.66.[16]

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PRESENTATION OF WALTER BRAZIE, M.D., FELLOWSHIP AWARD

Remarks made by doctor Walter Brazie, Kingman, Arizona, at the Annual Meeting of the Arizona Academy of Family Physicians in Scottsdale, February 18-21, 1976. The occasion was the presentation of the Walter Brazie, M.D. Award to Dr. Joyce Mobley for having been selected the oustanding Arizona Resident in Family Practice for the year 1975-76. This Fellowship Award was established in February, 1975, by members and friends of the Brazie family together with the Kingman Rotary Club and the Mohave County Medical Society.

Dr. Brazie closed his office in Kingman on March 31, 1976 after 51 years of practice in that community. At the age of 76 he looks well enough to serve another 51 years, but has been impelled to close his office because of the increase in malpractice insurance premiums.

"Dr. Mobley:

"You are entering your profession at a very critical time when we are fighting for our very existence. Many forces seemed to have combined to harass us.

"The politicians have produced a welfare system that has resulted in a generation growing up with a deep seated "something for nothing" philosophy. Ambulance chasing attorneys are encouraging people to blackmail insurance companies. Shortsighted insurance companies are making settlements out of court and increasing insurance premiums rather than contesting unwarranted claims. Non-thinking juries are being swayed to make preposterous awards based on emotion rather than fact.

"But, there is one other group who are contributing mightily to this crisis — I refer to our own Medical Profession. It is a paradox that when our Medical Schools are turning out the best educated and trained physicians in all our history that our public relations are at such a low ebb.

"To me the answer to the paradox is very simple. We have lost sight of the fact that medicine is an art as well as a science. Advances in the Science of Medicine have come faster in the past 25 years than in all the known history of Medicine. Demands upon Doctors are greater now than at any time in history. We have accomplished so many wonderful things that the public expects a miracle at every visit.

"How do we change the course of this sad state of affairs we find ourselves in? There is a simple answer to this dilemma — treat the patient first and then the disease.

"How do we know whether or not we are accomplishing this task? Again the answer is really simple. When your patients consult you about non-medical problems because they trust your honesty and judgment, you know you are making progress. When your patients upon leaving your consultation room say to the receptionist, 'I feel better already just talking to the doctor' — then you know you are getting the job done.

"It will be up to you people in Family Practice to point the way to practicing the Art as well as the Science of Medicine.

"I wish you the very best in all your future undertakings."

CLARENCE GRANT SALSBURY, M.D.

> You cannot choose your battlefield
> The Gods do that for you,
> But you can plant a standard,
> Where a standard never flew.
>
> Nathalia Crane

Few men, by their own efforts, good fortune and longevity, live to find themselves, living legends in their own time. But such is the good fortune of Doctor Salsbury.

A Canadian by origin, a medical missionary by choice, first in China 12 years; then to the Navajo Nation in Arizona 23 years; then medical administrator as commissioner of public health for the State of Arizona. At age 75 he accepted a part-time positoin as staff physician to the Infirmary at Arizona State University and a member of the Medical Review Team of the Arizona State Department of Welfare.

Nearly five hundred of his colleagues, friends, former patients, and most of all, many members of the Navajo Nation gathered in Phoenix at the Camelback Inn on May 22 to celebrate Doctor Salsbury's Ninetieth Birthday.

Seven States and two Provinces of Canada were represented at the luncheon. There were about 80 former students and co-workers from Ganado and about a dozen nurses who had graduated from the Sage Memorial School of Nursing. This was the first school to train Navajo Nurses and it was founded by Doctor Salsbury during his tenure at Ganado.

So, to this man of all seasons – Master Surgeon, Medical Missionary, Medical Administrator and benefactor to all those he served, to you Doctor Salsbury, your medical colleagues in Arizona extend to you our best wishes and congratulations on your Ninetieth.

HEALTH MAINTENANCE ASSOCIATES, LTD. RECEIVES NATIONAL ACCREDITATION

Health Maintenance Associates, Ltd. of Phoenix, Arizona has recently been notified of its accreditation by the American Group Practice Association.

As a result of a two-day on-site inspection by a team of physicians and administrators, it was determined that the level of care at Health Manitenance Associates, Ltd. meets and exceeds the standards of excellence outlined in the APGA Accreditation protoclo.

AGPA accreditation is a voluntary procedure. To qualify, a clinic must satisfy a professional survey team composed of experienced group practitioners. The clinic must meet a wide range of demanding criteria designed to assure the highest possible quality of patient care and diagnostic scope, medical educational activities, staff research and technological activities in support of medical practice. The quality of the staff and specialty distribution are considered, along with the organization, management, cost effectiveness and quality of the group's facilities. When the collective assessment of these factors meets AGPA standards, the group receives accreditation.

The AGPA is the national organization representing all forms of medical and dental group practices. It sponsors a variety of services and activities to encourage the maintenance and improvement of high quality health care delivery at reasonable cost to patients. The AGPA accreditation program is a primary means of pursuing this objective.

The AGPA accreditation protocol, developed in 1968, will serve as the basis for the newly formed Accreditation Council for Ambulatory Health Care, which, under the aegis of the Joint Commission on Accreditation of Hospitals, will assume all ambulatory care accreditation in April, 1976.

LACK OF REVENUE AND HIGHCOSTS TERMED THREAT TO SCIENCE JOURNALS

ASSOCIATED PRESS

WASHINGTON — The journals doctors read to keep abreast of new medical developments may be forced out of business because of rising costs, falling revenues and fewer members in academic groups, a Senate subcommittee was told recently.

A scientist whose company handles advertising for five major scientific journals testified the magazines face a threat that "could . . . produce a serious delay in . . . solving . . . many medical and scientific problems."

Earl J. Scherago, president of Scherago Associates Inc. of New York, said that without legistive assistance to control advertising in competing commercial magazines, and tax advantages for the scholarly magazines, the latter may become extinct.

Doctors generally receive the commercial magazines, supported heavily by drug company advertiisng, and the scholarly journals, carrying some drug advertising but also supported by academic societies.

Sherago blamed the medical communtiy for failing to support the nonprofit societies and their magazines.

He told a Senate Small Business subcommittee the drug companies are buying advertising space in magazines "which would cater to their demands of editorial and format control."

The scholarly journals, called "peer review journals" because the scientific articles must be approved by a panel of experts, generally have maintained strict policies prohibiting advertisers from influencing editorial content.

"Last year, as a group, 10 (commercial) publications, distributed to the research and laboratory field, collectively billed over $7 million in advertising revenue, money that would have been used to publish peer review material had those ads appeared in scolarly journals," Scherago said.

He said the nonprofit societies are increasing their dues to fight rising publishing costs.

Scherago said: "Learned journals often print adverse references to advertisers' products or present views which are unpopular with groups of advertisers. In no instance will a scholarly journal ever agree to run articles or product descriptions in exchange for advertising."

Among the magazines represented by Scherago is "Science," the largest "peer review journal." It is sent to the 140,000 members of the American Association for the Advancement of Science.

Letters to Editor

Editor
ARIZONA MEDICINE
Dear John:

In response to your editorial in the June issue of *Arizona Medicine* concerning the financial plight that the Arizona Medical Association finds itself in concerning the publishing of its medical journal I would like to make several comments and suggestions for ways to overcome the problem.

I believe that the future for Arizona's medical community is great. With the large increase in population expected by the year 2000, the medical facilities in this state will need to be expanded, further centralized and upgraded. Further, the increasing complexity that will be produced by this reorganization and the eventual entrance of the Federal government into our daily activities, will require a means to communicate these rapidly changing events. In addition, although there are many free "journals" being mailed to physicians, I think for the most part they are filled with advertising and limited helpful information for the practicing physician who is attempting to set aside some time for continuing medical education. I for one have made it a policy to file in the basket these journals without even perusing them. There just is not enough time to keep up with everything being published. I therefore feel that an additional role that *Arizona Medicine* could play would be to act as an instrument for continuing education for the physicians in this state.

In light of the above, I feel *Arizona Medicine* will come to play an important role in Arizona and feel strongly that it should be preserved at all costs. Although it presently costs approximately $50,000 a year to publish *Arizona Medicine* I think that we could streamline it somewhat to keep the costs down. For instance, the need for a shiny picturesque cover on every issue escapes me and I think a more practical one following the design of the *New England Journal of Medicine* would be more apropos. Furthermore, if no other area could be cut in the Association's budget to help subsidize the cost of the journal, then an additional source of revenue perhaps could be obtained by charging an additional $10.00 per year subscription fee which could be tacked onto the relicensure process. Although this is handled by the Board of Medical Examiners which is distinct from the Arizona Medical Association, perhaps an arrangement could be made between these two associations to allow this to take place. In return for fostering such a plan the State Board of Medical Examiners could have a ready format to present any information that they would desire to be distributed to the physicians in the State of Arizona. With approximately 6000 physicians licensed to practice medicine in the State of Arizona, this plan would bring in approximately $60,000 which would cover, I believe, the cost of publishing *Arizona Medicine*. Since we are providing a forum for the State Department of Health in our journal to present their point of view, an additional source of revenue could come from the appropriate state agency involved in health care within the state.

As a last resource, if all of the above fails, would be to combine *Arizona Medicine* with an existing regional medical journal. Such could be undertaken with either the *Rocky Mountain Medical Journal* or *Western Medicine*. I would hope, however, that *Arizona Medicine* can maintain its own identity.

I hope these comments will stimulate additional discourse concerning ways to continue publishing *Arizona Medicine*. It is, I believe, the most important aspect of continuing medical education that our Association supports.

Marshall B. Block, M.D.

Mr. Bruce Robinson
Managing Editor
ARIZONA MEDICINE
Dear Mr. Robinson:

Dr. John Vosskuhler's letter to the Editor of ARIZONA MEDICINE which appeared in the May 1976 issue needs an immediate response since it seems to have as its basis more of a concern for the needs of the radiological community than it does for the consumer public.

It is accepted and expected that orthopedists, chest physicians and cardiologists will perform and interpret their own x-ray "shadowgrams" because each holds the greatest expertise in his specialty in relating both the x-ray and clinical findings. It would now appear that the radiologist feels, somewhat presumptuously, that he should have as his exclusive professional domain the area of computed tomography of the nervous system. In reality, the nervous system is an area most radiologists, even when interpreting neurological "shadowgrams," have had but limited training and experience.

Computed tomography of the central nervous system no longer displays "shadowgrams" but instead images of neuroanatomy and neuropathology. Indeed, the original concept of this new and important neurological tool was the patented idea of a Neurologist (Oldendorf) and developed into its practical form by a computer engnieer (Hounsfield). The proliferation of this new technique in the hands of persons untrained in neurological disease and, therefore, ill equipped to interpret these computed display images has seriously concerned neurologists and neurosurgeons. In December 1975 the American Academy of Neurology and the American Neurological Association issued the following joint statement:

"Because Neurologists have traditionally been concerned with neurodiagnostic procedures and their interpretation, because the advent of new techniques for imaging the brain and spinal cord, for measuring blood flow and neural function, are of greatest importance for care of patients with neurologic disorders and for teaching and research, therefore the Association wish to emphasize that neurologists must be involved in the decision making, performance, and interpretation of these procedures. Neurologists must also be intimately involved with planning, implementation, and execution of programs designed to teach these new techniques to physicians, technicians, and students."

In April 1976 the American Association of Neurological Surgeons adopted an identical resolution with the insertion of the words "neurologists and neurosurgeons" wherever the word "neurologists" was used in the original statement.

On April 30, 1976 the membership of the American Academy of Neurology adopted a series of supplementary resolutions intended for implementation of the above statement of principle. Two of these supplementary resolutions are of particular import to the consumer public for the effective utilization of computed tomographic scanning of the nervous system.

"1. Establish criteria for quality control and utilization.

2. Specify that only specialists trained in neurological disorders are qualified to interpret computerized tomography of the nervous system. Interpretation requires involvement in management of the laboratory and performance of the test."

Obviously, training and experience in another specialty, and specifically in radiology, does not by itself produce competence in this new area of imaging. This is particularly true where in computed tomography of the nervous system an understanding of neuro-anatomy, neuropathology and clinical neurological disease is of prime importance in obtaining adequate and accurate interpretation of the test images. We would hope that physicians be qualified in the above paramaters before establishing such laboratories and before attempting interpretation of these new forms of imaging.

The neurologist's concern is for the quality with which computed neurological scans are performed and in the quality of their interpretation. By excellence in these parameters, patients with neurological disease will obtain the best possible neurological care available in the State of Arizona. We would hope that this would be the ultimate goal of all physicians irrespective of specialty.

Sincerely,
ALAN YUDELL, M.D.
Practice Committee and
Imaging Committee
American Academy of Neurology

Editor, Arizona Medicine
Phoenix, Arizona

Sir:

Five years ago considerable effort was expended trying to get a medical director at St. Joseph's Hospital. The medical associations and doctors from other hospital staffs fought this adamantly — preaching hell fire and brimstone.

As of 1 April 1976 Good Samaritan Hospital installs a medical director without debate or comment.

Yes, Virginia, there is a double set of standards!

William H. Harper, M.D.

EDITORS NOTE: For those who wish more controversial subjects, this should sweep the high mesas! Now lets hear your opinions!

6 April, 1976

Dr. John W. Kennedy,
Editor, Arizona Medicine,

Dear Dr. Kennedy:

I am writing to tell you how much I enjoy the historical items which are found in Arizona Medicine, and to offer a correction for one small error found in the quiz section of the March issue where your reference has doubtless supplied you with misinformation. I refer to Assistant Surgeon B.J.D. Irwin, Medical Inspector of General Nelson's division of the Army of the Ohio who was commended for his work at the battle of Shiloh, not because he organized a large hospital — actually it contained only three hundred beds — but because it was the first tent field hospital, a type of facility which was soon found to be superior to all other types because of the diminished incidence of erysipelas and hospital gangrene among the wounded when they were cared for in such installations.

In 1862 there was no provision in Army Regulations for the promotion of medical officers, or for increasing their pay or for providing any other reward for duties well done, so it was gratifying to learn that Dr. Irwin continued to perform brilliantly after the war, and that he was finally awarded the Medal of Honor. The commendation which his commanding officer wrote for him after the battle of Shiloh was the only one, of which I have any knowledge, written in behalf of a medical officer. It follows:

1 Col. J. B. Fry, Asst. Adjutant General,
Chief of Staff.

"Sir, I desire to call the especial attention of the General commanding this Army to the desirable hospital accommodations adopted by Dr. Irwin, U.S.A., Medical Inspector of the Division, during the battle of the 7th inst. (April 7, 1862). They will be found as nearly perfect as the circumstances would allow. The founded in the Division did not suffer from the inclement weather; they were all promptly sheltered and cared for. I beg to recommend Dr. Irwin to the favorable consideration of his superiors.

Very respectfully,
W. Nelson, Brigadier General

Surgeon R. Murray, U.S.A., Medical Director of the Army of the Ohio, wrote briefly of Dr. Irwin's ingenuity in his report to the Surgeon General.

2

"Assistant Surgeon Irwin deserves very great credit for his admirable management on the field, as well as for his promptness and professional skill in the care of the wounded. He found a house on the left of the field, where his Division was engaged, and, by securing tents from the neighboring camp, and collecting camp furniture, he succeeded in organizing and putting in good working order a hospital for 300 patients, partly of the wounded of his own command, partly of those of General Grant's Army, who were wounded in that part of the field, and partly of Confederate wounded. These were so well taken care of in every way, that I designated them as the last to be sent off on board of the boats." (evacuation by river boats) The house was used as an operating room and kitchen, the tents for wards.

So it was good to meet Assistant Surgeon B.J.D. Irwin, U.S.A., again, and to learn of his belated award. Being a recent Arizonan, may I ask; where is Apache Pass and when did that action take place? Was Surgeon Irwin killed there?

EDITORS NOTE — Brigadier General B.J.D. Irwin died in his bed age 87, December 15, 1917 and is buried at West Point, New York.

He was recommended for the medal of Honor for his service with the infantry serving at Fort Buchanan, A.Z.T. The action was February 11, 1860, the citation read "Awarded the Medal of Honor for distinguished gallantry in action against Chiricualma Apache Indians, near Apache Pass, Arizona February 13-14, 1816, while serving as assistant surgeon —."

If we are correctly informed he was also the first medical officer of the U. S. Army to receive this Medal of Honor.

ArMA Medical History

THE MAKING OF THE HOSPITAL IN WINSLOW ARIZONA

H. S. BECKWITH, M.D.

Winslow, a small metropolis in Northern Arizona was without a hospital until 1936 when an adobe structure was built by patients of Dr. Robert Stump endeavoring to pay off their medical and surgical costs, they had incurred.

Here we interpose information supplied by Joyce Stump Johnson, Dr. Stump's daughter. She wrote in part, "What can I tell you about the hospital in Winslow — not much but I do remember the building of it and all of the labor; love and fun that went into it. I was too small to remember all of the many details but I do recall having the run of the place, washing dirty baby diapers and other laundry, folding numerous materials for sterilization and other small things a child could and did do. I was only two when my father moved to Winslow and I was all of eleven when we left and I remember a lot of things that won't make a newspaper article." Dr. Robert Stump added, "I went to Winslow in 1928 to work for the Santa Fe railroad. They had a small hospital but it was not used as such. The nearest hospitals were in Flagstaff, sixty-five miles to the west or in Gallup, New Mexico, one hundred twenty-five miles to the east. The need of a hospital became very apparent — accidents involving the railroad personnel, and even in those days a considerable amount of traffic accidents on highway 66 the main street of the town. Not only did I do all my own x-rays (yes that accounts for the burns on both hands) but all major surgery from gallbladders to C-sections. I sold my hospital to Dr. M. G. Wright when I left Winslow and moved to Phoenix in 1936." He further continues, "The hospital was built just as Dr. Beckwith describes, patients who could exchange their talents in the building lines in helping to build the hospital and paid off medical, obstretical and surgical costs at the same time."

Now we pick up Dr. Beckwith's account again.

Prior to the arrival of Dr. Stump, Mrs. Frances Stevenson, a registered nurse, decided about the time of World War I to consolidate maternity care and delivery into a single structure instead of out in individual homes. She being a graduate registered nurse saw the need for a central place for patient care. Her first venture was a maternity ward in the Mexican American part of town. In the late 1930's she cared for premature babies in a homemade incubator. Several years later she moved the maternity home from the Mexican American section to a home on the north side of the railroad tracks where she took all nationalities. The house was located on East Aspinwall and was remodeled to better accommodate a maternity hospital.

In 1945, Maude Miller, also a graduate registered nurse, joined her sister and together they leased the building on West Second Street and Hicks which Dr. M. G. Wright had been using for surgery and care of his individual patients. Up until this time there had been no facilities for doctors in this town to hospitalize their patients. Frances Stevenson and Maude Miller together managed the hospital.

With the return of physicians from the Armed Forces following WW II they were very much aware of the necessity for a more adequate and better equipped hospital than was available at the time. So, in 1951 with a strong community effort, the citizens of Winslow formed a non-profit corporation. Funds for the new hospital were collected by selling shares, each person donating money and became a stockholder which gave them voting privileges. This new 24 bed hospital was opened in 1954. After considerable effort in shaving expenses, it was built at a cost of $375,000, half of which was from a Hill-Burton grant. All services were provided

except for certain specialized types of surgery. In 1958 an addition was built to the hospital raising the patient count to 31. At this time there was approximately four or five doctors working in and out of the hospital. By 1965, the hospital had outgrown this building and since it was impossible to enlarge it, plans were started to build a new hospital.

Winslow Memorial Hospital moved to 1501 Williamson Avenue in October 1971. The present hospital is a 42 bed hospital, fully equipped with two surgeries, one delivery room, one labor room, a complete emergency room, intensive care unit, four beds designated as CCU and ICU beds, and is well staffed with nurses and aids who adequately attend the patients. The building is so constructed that another addition of 42 beds could be added if the need arises. At present the hospital is staffed by one general surgeon and four general practitioners dedicated to family practice. The hospital is suffering from the exorbitant costs for medical and surgical supplies and personnel salaries.

MEMORIAL HOSPITAL HISTORY

By BARBARA LAMBESIS,
Director of Community Affairs

Early in 1934, the cruel effects of the Great Depression were keenly felt in the community of South Phoenix. The residents had few utilities and pitifully inadequate housing. In addition, medical faciliteis were scarce with only two hospitals serving the entire Phoenix area. Hope for a better life for the residents seemed to vanish as the Depression lingered.

During 1934, St. Monica's Community Center was established in South Phoenix. It was housed in an unused grocery store on the corner of South Seventh Avenue and Sherman Street.

St. Monica's Center was the beginning of the realization of the dreams and visions of its founder, Emmett McLoughlin who, until his death in 1970, worked to bring hospital services, housing and hope to the sick and deprived of South Phoenix. He was supported and encouraged by those who saw the same visions, shared the same dreams, and worked with the same dedication toward fulfilling the needs of all people regardless of race, creed or economic status.

St. Monica's Center was the first in the state to establish an out-patient prenatal clinic. An old barber shop was converted into a clinic building and with the cooperation of Dr. Preston Brown and Sister Mary Monica of St. Joseph's Hospital of Phoenix, a maternity service was initiated.

The Maternity service inspired the addition of other health care services at St. Monica's Community Center. Soon a "well-baby" clinic and a venereal disease clinic were established with the help of the United States Public Health Service which arranged for the transfer of a nurse to St. Monica's from Fort Huachuca.

Within a short time, the prenatal clinic and delivery service became overloaded and could no longer handle all the maternity cases. The only solution was to build an obstetrical facility at the Community Center. A 12 x 24 foot prefabricated building was donated by the O'Malley Lumber Company and erected one Sunday by

In 1934, these buildings housed St. Monica's Community Center . . . the beginning of Memorial Hospital.

the men of the neighborhood. Hundreds of babies were born in that humble frame building which was later to inspire the construction of a hospital.

During the Depression, St. Monica's Community Center tried to meet not only the health care needs of the people living in South Phoenix, but also established a food distribution center, day nursery, sports center, and a sewing club where women made quilts and clothes from scraps of fabric acquired from all over town.

Times began to change however, and by 1940 the profile of Phoenix was changing too. Luke Air Field and Williams Air Force Base were in operation. The Reynolds Aluminum plant had been built and the Defense Program had more than doubled the population of Phoenix. The two hospitals in the Valley became very crowded, but adequate health care facilities were still out of reach for many citizens of South Phoenix.

About this time the Board of Directors of St. Monica's Community Center decided to apply to the United States Public Health Service for the resources required for a new community hospital.

Emmett McLoughlin and the Board of St. Monica's had already been successful in bringing about the construction of three federally funded housing projects, Marcos de Niza, Matthew Hensen and Duppa Villa. Families that once inhabited one room shanties and children that once suffered from cold winds sweeping through cardboard walls were provided clean and comfortable quarters. If housing could be built, why not a hospital? But the application to the United States Public Health Service came at a bad time and a miracle was in order.

The application arrived in Washington only a few days before the attack on Pearl Harbor. The United States entered World War II and the War Production Board was established immediately to conserve strategic materials needed in the war effort. Senators Carl Hayden and Ernest McFarland of Arizona worked tirelessly for over a year to convince the military and the War Production Board of the need for the hospital. The application came before the War Production Board and was rejected six consecutive times. The seventh time it was approved.

The hospital was built by the Federal Works Agency on property owned by St. Monica's. A total of $9000 was raised through newspaper subscriptions, contests, carnivals, and bazaars in an effort to purchase 14 additional acres of land. A Board of Directors organized the St. Monica's

Emmett McLoughlin, first administrator of St. Monica's Hospital, presided over groundbreaking ceremonies for the Hospital.

This picture, taken in 1943, shows the beginning of construction of St. Monica's Hospital and Health Care Center.

This aerial view of St. Monica's hospital and Health Care Center was taken in 1945. The name was changed to Memorial Hospital in 1949 to honor those who had died in World War II.

Hospital and Health Center Corporation which leased the hospital plant from the Federal Works Agency. The hospital opened its doors on February 14, 1944, as a private, non-profit institution. In July 1947, St. Monica's Hospital was sold by the Federal Government to the Board of Directors for $200,000 – millions of dollars less than its actual value.

St. Monica's received national recognition when it initiated the first racially integrated school of nursing west of the Mississippi. The first class of nurses began training in October 1944, shortly after the hospital opened. The United States Public Health Service approved designation of St. Monica's Nursing School as a United States Nurse Corps Training Center and granted the school a budget of $41,955. The first class of nurses numbered 37 and about half the students were American Indian, Black, Mexican/American, or Oriental.

Eleanor Roosevelt, who had taken a personal interest in the project, visited the nursing school and presented an address before the student body. She noted the school was one of the first in the nation to defy racial prejudice and was a monument to the courage, vision, and loyalty of its founder and the principles of brotherly love.

Over the years, some 15,000 children were examined and treated at Memorial's Children's Clinic.

St. Monica's Hospital Will Graduate 21

St. Monica's received national recognition when it initiated the first racially integrated school of nursing west of the Mississippi.

In 1949, the Hospital's name was changed to Humanity Memorial Hospital in honor of those who had died in the war. Later the name was shortened to Memorial Hospital.

Memorial Hospital began as an out-patient facility and as it developed toward a comprehensive hospital and medical center, it continued to emphasize out-patient services.

The first out-patient service to be established was the Children's Clinic under the direction of Dr. Trevor Browne, a Phoenix city school physician. Dr. Browne inspired the formation of the first working ladies' auxiliary in the state. The Auxiliary assisted the Hospital through fund-raising activities and by helping staff the Children's Clinic. Over the years, some 15,000 children in the Phoenix area were examined and treated at the Clinic at no charge. As far as it is known, the Children's Clinic was the only all-inclusive, city-wide, private children's clinic in the United States at the time.

Other clinics housed at Memorial in its early years and still in operation include Planned Parenthood, the Elk's Dental Clinic, the Cancer Clinic and the Maternity Services Clinic. Today, nearly thirty percent of the Hospital's medical care service program is attributed to out-patient visits.

Memorial was the first in Phoenix to develop services for patients afflicted with polio. It had 70 iron lungs and patients came from all neighboring states.

In 1961, Memorial became the first in the state to have a cancer research program complete with cobalt therapy, nuclear laboratory and experimental chemotherapy program. The first regional perfusion and continuous infusion procedures were performed at Memorial.

In the mid and late 1960's the Hospital began to experience some difficult times. Dreams, visions, and miracles were no longer substitutes for sound management practices in the increasingly complex structure of health care delivery. Even so, the Board of Trustees continued its commitment to the development of services for the greater South Mountain area of Phoenix. The need for senior citizen housing was more than apparent and again it was the Hospital that played an important role in providing for one of man's basic needs — housing.

In 1964, two eleven-story buildings, housing 153 apartments for senior citizens, were built on hospital property. The Towers, as they are known, provide comfortable living and medical security for residents in their "golden years." The Towers became a Phoenix landmark in 1973 when a bright geometric design was painted on the grey cement walls. The painted walls of the Towers can be seen clearly for miles around and have become symbolic of the turn-around the hospital has made during the past few years.

Activity and demand for services have in-

Memorial was the first in the state to develop a cancer research program. Here Dr. Frank Tolone radiologist, and two nurses examine cobalt equipment.

creased dramatically and economic indicators demonstrate a sound future.

The Hospital provides services for patients residing throughout Metropolitan Phoenix, Maricopa County and Arizona. It boasts a unique nurse-midwife and family-centered childbirth program, a strong cancer program and an excellent orthopaedic department serving such organizations as the Phoenix Suns and the Sun Devils of Arizona State University. In addition, Memorial provides all the other services normally found at a comprehensive hospital/medical center. The staff of Phoenix Memorial is dedicated and enthusiastic — always looking to the future.

The Memorial Towers, built in 1964 to meet the housing needs of senior citizens, were painted with a bright geometric design in 1973 — a year of rebirth for Memorial.

Memorial Hospital's Long Range Master Plan calls for a most enterprising expansion and building replacement program. The first phase calls for a multi-million dollar construction project to begin in mid 1976.

A free-standing 44-suite medical office building will be constructed on ground adjacent to the hospital. In addition, the Board of Trustees has authorized the administration of Memorial Hospital to proceed with plans for the construction of ambulatory care centers in South Phoenix.

Currently, the President and administrator of Phoenix Memorial is Reginald M. Ballantyne III. The chairman of the Board of Trustees is Augustus Shaw.

Human compassion lead to the creation of Memorial Hospital which grew with the dreams and visions of those whose dedication to humanity lead them to build not just a facility for healing, but a symbol of man's responsibility to his fellow man. Memorial Hospital — its heritage is noble and its future is bright.

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

EXECUTIVE COMMITTEE

The Executive Committee meeting of the Arizona Medical Association, Inc. held in the Lanai Room, Safari Hotel, Scottsdale, AZ on Tuesday, April 27, 1976 convened at 7:08 a.m., William C. Scott, M.D., President and Chairman, presiding.

MINUTES

The minutes of the meeting held March 19, 1976, were approved as distributed.

MALPRACTICE ASSESSMENT

Non-Payment

The following have not paid the assessment as of 4/20/76 and have been advised that they have been suspended:

1. Paul G. Bauer, M.D. — Maricopa
2. Milton M. Connell, M.D. — Maricopa
3. Earl S. Cronk, M.D. — Maricopa
4. A. Howard Erickson, M.D. — Maricopa
5. Eldon B. Hamblin, M.D. — Maricopa
6. William H. Harper, M.D. — Maricopa
7. Noah M. Koenigsberg, M.D. — Maricopa
8. Jack L. Loudermilk, M.D. — Maricopa
9. Sterling J. Ryerson, M.D. — Maricopa
10. Tadeausz Zakrzewski, M.D. — Maricopa
11. Hugh B. Allen, M.D. — Pima
12. Leonard C. Baldauf, M.D. — Pima
13. Larry J. Buckels, M.D. — Pima
14. Ian M. Chester, M.D. — Pima
15. Michael W. Cohen, M.D. — Pima
16. James J. Corrigan, Jr., M.D. — Pima
17. Benjamin T. Edwards, M.D. — Pima
18. Vincent A. Fulginiti, M.D. — Pima
19. Ronald C. Hansen, M.D. — Pima
20. Thomas R. Harris, M.D. — Pima
21. Charles E. Harter, M.D. — Pima
22. Elmer S. Lightner, M.D. — Pima
23. Thomas E. Newman, M.D. — Pima
24. Loyd C. Planting, M.D. — Pima
25. C. Jack Snider, M.D. — Pima
26. Anita M. Stafford, M.D. — Pima
27. Paula L. Stillman, M.D. — Pima
28. Lynn M. Taussig, M.D. — Pima
29. Willard R. Van Nostrand, M.D. — Pima
30. Anacieto C. Veloso, M.D. — Pima
31. John K. Wenham, M.D. — Pima
32. Kenneth F. Conry, M.D. — Mohave
33. Robert L. Hyde, M.D. — Pinal
34. Jeanne B. Johnson, M.D. — Yuma

Requests for Exemption

It was moved and carried to grant the requested exemptions to the following due to financial hardship:

1. Hugh B. Allen, M.D. — Pima
2. Michael W. Cohen, M.D. — Pima
3. James J. Corrigan, M.D. — Pima
4. Vincent A. Fulginiti, M.D. — Pima
5. Ronald C. Hansen, M.D. — Pima
6. Thomas R. Harris, M.D. — Pima
7. Elmer S. Lightner, M.D. — Pima
8. Thomas E. Newman, M.D. — Pima
9. Anita M. Stafford, M.D. — Pima
10. Paula L. Stillman, M.D. — Pima
11. Lynn M. Taussig, M.D. — Pima

1976-77 MEETING SCHEDULE

The following meeting schedule for the Executive Committee was approved:

May 22, 1976 — Saturday — Special with county society presidents and wives
June 18, 1976 — Friday
July 16, 1976 — Friday
August 13, 1976 — Friday
September 17, 1976 — Friday
October 15, 1976 — Friday
November 19, 1976 — Friday
December 17, 1976 — Friday
January 21, 1977 — Friday
February 18, 1977 — Friday
March 18, 1977 — Friday
April 26, 1977 — Tuesday — Annual Meeting

A. O. M. A.

The letter from L. George Hersey, D.O., president of the A. O. M. A. regarding reciprocating at each other's board meetings was received.

CONSULTATE OF MEXICO

The letter of 3/26/76 from Rene L. Morlet, Counsel of Mexico regarding limitations on American physicians practicing in Mexico was reviewed. It was determined to convey the essence of the letter in a subsequent MEDICAL MEMO.

EDITOR-IN-CHIEF

Dr. Kennedy's letter of 1/7/76 regarding additional items for ARIZONA MEDICINE was received.

RESOLUTION 23-76

It was moved and carried to introduce Resolution 23-76 "recognition of the efforts of the Governor and the 32nd Legislature" to the House of Delegates.

RESOLUTION 24-76

It was moved and carried to introduce Resolution 24-76 "Continuation of Malpractice Insurance Crisis Committee and Special Assessment" to the House of Delegates.

REVIEW OF BOARD OF DIRECTORS AGENDA

The Committee reviewed the Board of Directors agenda for 4/27/76 and made various recommendations.

The record must show that William G. Payne, M.D. did not participate in the discussion relating to Agenda Items 5A, B, C, & D.

DEPARTMENT OF HEALTH SERVICES

Dr. Danboy brought the committee up to date regarding plans for the swine influenza immunization program.

Meeting adjourned 8:35 a.m.

William E. Crisp, M.D.
Secretary

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc. held in the Embassy Room of the Safari Hotel, Scottsdale, AZ on Tuesday, April 27, 1976, a quorum being present, convened at 9:04 a.m., William C. Scott, M.D., president and chairman, presiding.

WELCOME

Dr. Scott welcomed the several county society presidents and other guests.

MINUTES

The minutes of the Board of Directors meeting of 2/21/76 and 3/7/76 were approved as distrbiuted.

ArMPAC

Dr. Langston reported that membership was down from last year but is gradually growing. He also reviewed Langston invited all members to the ArMPAC banquet on Thursday, April 29, 1976.

BOARD OF DIRECTORS

BOMEX Lease

It was moved and carried to approve the lease with the Board of Medical Examiners for the period 7/1/76 thru 6/30/77 with an option for an additional year at the rate of $6.00 per square foot.

BOMEX Appointment

It was moved and carried to recommend to the Governor that Richard L. Dexter, M.D. be reappointed to the Board of Medical Examiners for the term 7/1/76 thru 6/30/81. The Executive Committee is also authorized to submit additional names if appropriate.

Resolution 18-76

It was moved and carried to introduce the following resolution to the House of Delegates.

SUBJECT: RATIFICATION OF LOAN

WHEREAS, The Board of Directors of the corporation on February 7, 1976 and on March 7, 1976, duly authorized the borrowing of $1,000,000 from The Valley National Bank of Arizona and First National Bank of Arizona for the purpose of advancing such funds to Mutual Insurance Company of Arizona ("MICA") in order to provide MICA with its initial capital requirements, and

WHEREAS, The borrowing by the corporation and the advance to MICA were completed by the officers of the corporation on March 18, 1976, and

WHEREAS, It has been suggested that the members of the House of Delegates approve and ratify such borrowing and advance, therefore be it,

RESOLVED, That the borrowing of $1,000,000 by the corporation from The Valley National Bank of Arizona and First National Bank of Arizona and the advance of such funds to MICA for the purposes of providing its initial capital be and the same is hereby ratified and approved by the House of Delegates.

Resolution A-4-76

It was moved and carried to introduce the following resolution to the House of Delegates.

SUBJECT: UPDATING ARTICLES OF INCORPORATION

WHEREAS, The Arizona corporation laws have been amended, effective July 1, 1976, and this corporation desires to take advantage of such new corporation laws, therefore be it,

RESOLVED, That Article IV of the corporation's Articles of incorporaiton be manded to read:

ARTICLE IV

The time for the commencement of this corporation shall be the date of the filing of these Articles of Incorporation as required by law and the term of its corporate existence shall be twenty-five (25) years [thereafter] AFTER JUNE 19, 1975, with privilege of renewal as provided by law, PROVIDED THAT SUCH TWENTY-FIVE (25) YEAR LIMITATION SHALL NOT APPLY IF AND WHEN IT IS NO LONGER REQUIRED BY ARIZONA LAW, IN WHICH CASE THE EXISTENCE OF THE CORPORATION SHALL BE PERPETUAL.

and, be it further

RESOLVED, That Article XII of the corporation's Articles of Incorporation be amended to read:

ARTICLE XII

The highest amount of indebtedness or liability direct or contingent, to which this corporation may be subject at any time shall be such amount or amounts as may be authorized by three-fourths (3/4) of the votes cast with respect thereto at a lawfully held meeting of the members of the House of Delegates and approved by the Arizona Corporation Commission, PROVIDED THAT THIS LIMITATION ON INDEBTEDNESS OR LIABILITY SHALL BE OF NO FURTHER FORCE OR EFFECT WHATSOEVER IF AND WHEN IT IS NO LONGER REQUIRED BY ARIZONA LAW.

and, be it further

RESOLVED, That a new Article XIV be added to the corporation's Articles of incorporation, to be as follows:

ARTICLE XIV

THE CORPORATION MAY INDEMNIFY AND AND ALL OF ITS PRESENT OR FORMER DIRECTORS, OFFICERS, EMPLOYEES, OR AGENTS TO THE MAXIMUM EXTENT PERMITTED BY APPLICABLE LAW. WITHOUT LIMITING THE GENERALITY OF THE FOREGOING, THE CORPORATION MAY INDEMNIFY AND AND ALL OF ITS DIRECTORS AND OFFICERS, OR FORMER DIRECTORS AND OFFICERS, AGAINST EXPENSES INCURRED BY THEM, INCLUDING LEGAL FEES, OR JUDGMENTS OR PENALTIES RENDERED OR LEVIED AGAINST ANY SUCH PERSON IN A LEGAL ACTION BROUGHT AGAINST ANY SUCH PERSON FOR ACTIONS OR OMISSIONS ALLEGED TO HAVE BEEN COMMITTED BY ANY SUCH PERSON WHILE ACTING WITHIN THE SCOPE OF HIS EMPLOYMENT AS A DIRECTOR OR OFFICER OF THE CORPORATION, PROVIDED THAT THE BOARD OF DIRECTORS SHALL DETERMINE IN GOOD FAITH THAT SUCH PERSON DID NOT ACT, FAIL TO ACT, OR REFUSE TO ACT WILFULLY OR WITH GROSS NEGLIGENCE OR WITH FRAUDULENT CRIMINAL INTENT IN REGARD TO THE MATTER INVOLVED IN THE ACTION.

Note: All bracketed, bald face words represent deletions; all fully capitalized words are new material. These resolutions must be approved by three-fourths of the votes cast thereon.

Nominee for Blue Cross Board of Directors

It was moved and carried to approve Walter K. Grigg, M.D. as a nominee to replace Wilfred Potter, M.D. on the Board of Directors of Arizona Blue Cross.

1976-77 Meeting Schedule

It was moved and carried to adopt the following meeting schedule:

June 12, 1976 — Saturday
September 18, 1976 — Saturday
November 20, 1976 — Saturday
February 26, 1977 — Saturday
April 26, 1977 — Tuesday, Annual Meeting
April 30, 197 — Saturday, Annual Meeting

EXECUTIVE COMMITTEE

Classification Changes Approved

(1) Maricopa County Medical Society
  (a) Roy O. Bjork, M.D. — Active to Active Over 70—Account Age — Dues Exempt Effective 1/1/76
  (b) Jean Carnes, M.D. — Active to Associate — Account Residency — Effective 7/1/76
  (c) Robert B. Ellis, M.D. — Active to Active Over 70 — Account Age — Dues Exempt Effective 1/1/76
(2) Mohave County Medical Society
  (a) Mel J. Harvey, M.D. — Active to Associate — Account Residency — Effective 1/1/76
(3) Pima County Medical Society
  (a) Virginia S. Furrow, M.D. — Active to Associate — Account Retirement — Dues Exempt Effective 1/1/76
  (b) A. Harry Neffson, M.D. — Active to Associate — Account Retirement — Dues Exempt Effective 1/1/76
  (c) Louis E. Young, M.D. — Active to Associate — Account Illness — Dues Exempt — Effective 1/1/76
(4) Santa Cruz County Medical Society
  (a) Zenas B. Noon, M.D. — Active to Active Over 70 — Account Age — Dues Exempt Effective 1/1/76
(5) Yuma County Medical Society
  (a) A. J. Ochsner, M.D. — Active to Associate — Account Retirement—Dues Exempt Effective 1/1/76
  (b) James Volpe, M.D, — Active to Associate — Account Retirement—Dues Exempt Effective 1/1/76

Dues Exemption Requests Approved

(1) Maricopa County Medical Society
  (a) Lois McLean, M.D. — financial hardship
  (b) C. James Statt, M.D. — financial hardship

FINANCE COMMITTEE

Dr. Dexter reviewed the financial statement for the period ending 3/31/76 — RECEIVED.

GRIEVANCE COMMITTEE

The matter of the conflict at the University of Arizona was discussed at length reviewing the Pima County Medical Society Board of Mediation report, Gordon R. Tobin, M.D.'s letter of April 3, 1976, the recommendation of the Housestaff Section dated 4/16/76 and a letter dated April 17, 1976 signed by 28 physicians. The following actions were taken:

Please note that William G. Payne, M.D. and William C. Scott, M.D. did not participate in the discussion nor voting. Richard L. Dexter, M.D. abstained from voting.

It was moved and carried that the Association write to the Board of Regents stating that the Association has received certain documents, which are to be included with the letter, from the Pima County Medical Society concerning the Department of Surgery at the University of Arizona College of Medicine. Further stating that the Association would be willing to act as a resource body for the Board of Regents by investigating the matters contained in these materials if the Board of Regents so requests.

It was moved and carried to send the Pima County Medical Society material to the American Medical Association with a statement that there appears to be substance to the report and that we recommend that the American Medical Association develop an adequate and effective model grievance procedure for Housestaff and Physician Faculty. That this matter also be referred to the Grievance Committee of the Arizona Medical Association for review and appropriate action.

HEALTH MANPOWER COMMITTEE

Arizona Health Service Corp.

HEW's letter dated 4/12/76 advising us that the Arizona Health Service Corp. proposal had been disapproved was received.

MALPRACTICE INSURANCE CRISIS COMMITTEE

Letters from Mark I. Harrison, President of the Arizona Bar Association, William B. Robey, M.D. and Merrill S. Chernov, M.D. were received for information.

CORRESPONDENCE

Robert J. Oliver, M.D.'s letter of 3/22/76 congratulating the Association on behalf of the Tucson Awareness House, for its position on decriminalization of marjuana laws was received for information.

OTHER BUSINESS

Brookdale Award

No nominations were made.

National Association of Blue Shield Plans Task Force Report

Mr. Jacobson, legal counsel, reviewed the legal aspects of the relationship between ArMA and Arizona Blue Shield. It was his recommendation that the Association seriously consider disassociating itself from Arizona Blue Shield. No action was taken as this is properly a matter for the House of Delegates and the Blue Shield Corporate Body.

Housestaff Section

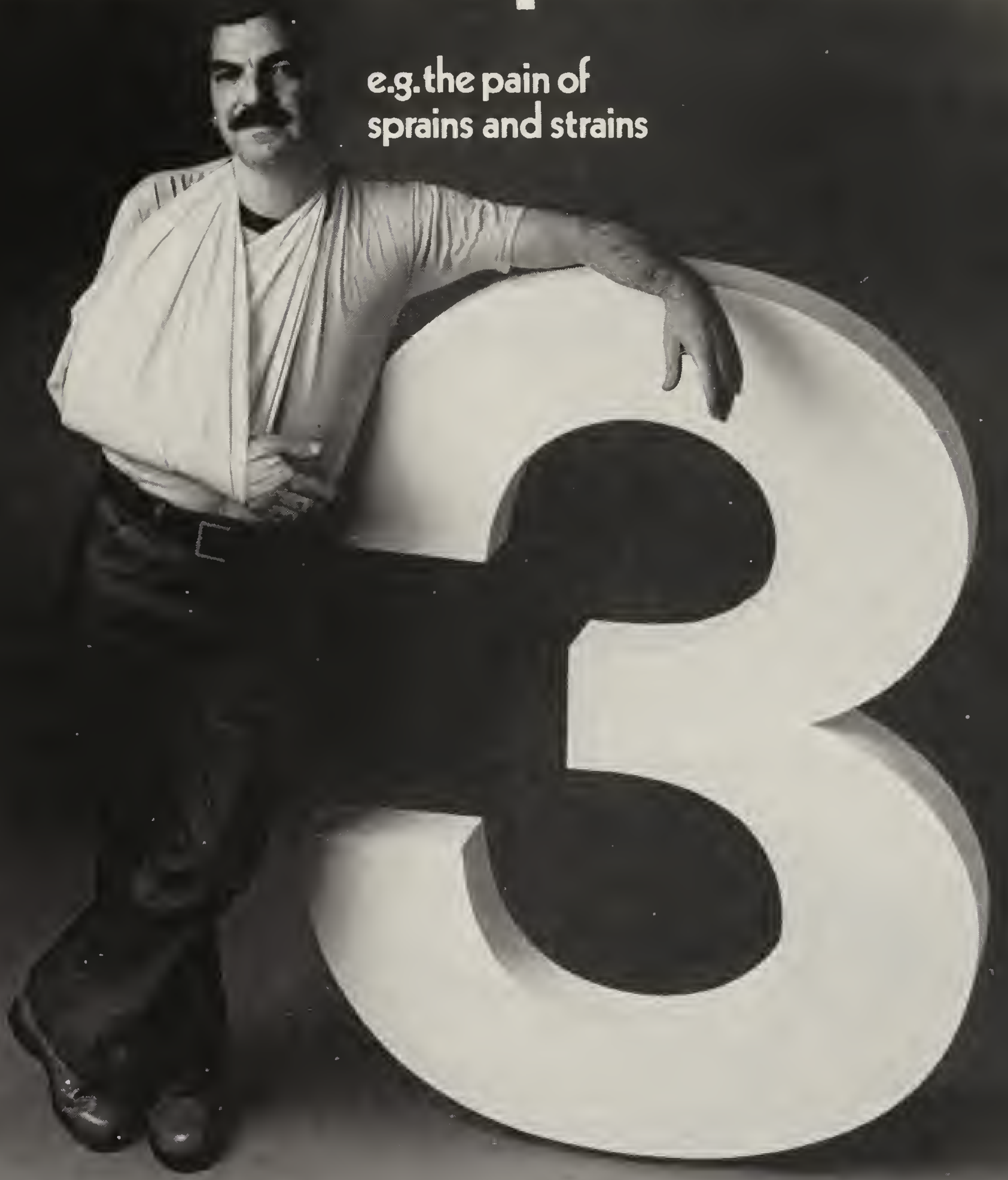
It was moved and carried to approve the finances to send two members of the Housestaff Section to the AMA Annual Meeting in Dallas in June. At subsequent meetings we are to revert to the original policy of sending only one Housestaff Section member to AMA meeting.

Meeting adjourned 10:55 a.m.

William E. Crisp, M.D.
Secretary

No. 3
As potent as the pain it relieves.

e.g. the pain of
sprains and strains



NOT TOO LITTLE
- as potent as the pain you need to relieve in patients with fractures, sprains, strains, wounds, contusions, and the pain of surgical convalescence
- unlike acetaminophen/codeine combinations, it does not sacrifice anti-inflammatory action

NOT TOO MUCH
- potent—yet not excessive
- addiction liability low

NOT TOO EXPENSIVE
- brand-name quality, yet reasonable in cost
- readily available in both hospital and local pharmacies

Ⓒ III CONVENIENCE
- telephone Rx in most states, up to 5 refills in 6 months at your discretion (where state law permits)

EMPIRIN® COMPOUND
WITH CODEINE NO. 3

codeine phosphate* (32.4 mg) gr ½
Each tablet also contains: aspirin gr 3½, phenacetin gr 2½, caffeine gr ½. *Warning–may be habit-forming

Wellcome
Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

"Joint Life is the insurance of the '70's"

Doctor, HBA Life's

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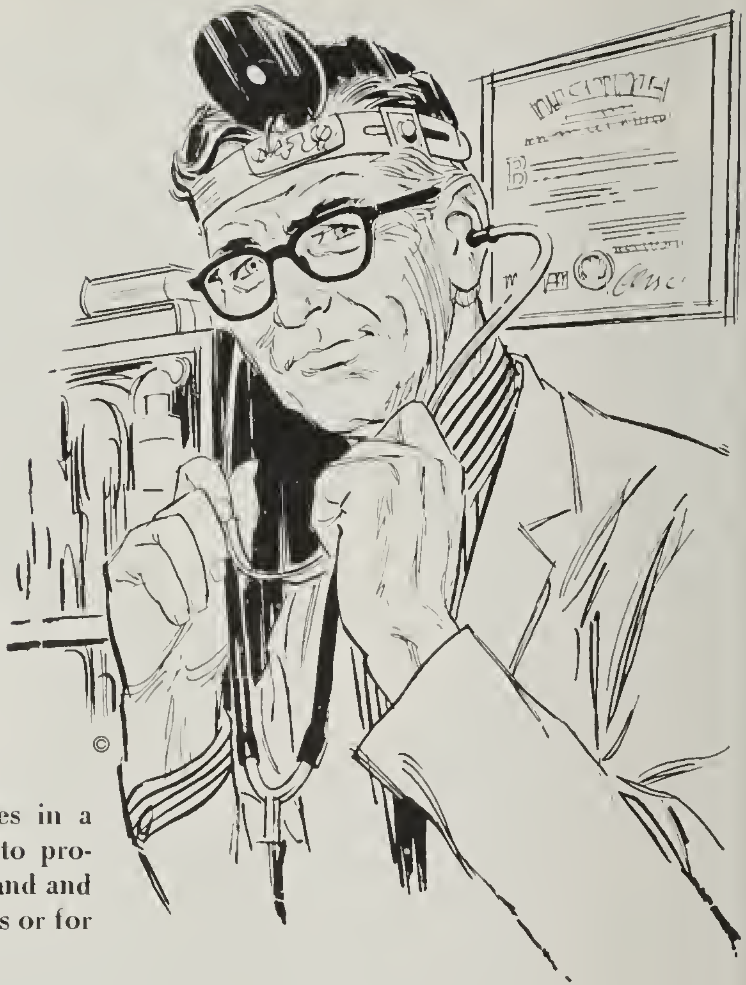

BUSINESS INSURANCE: Two doctors, partners or associates in a professional corporation, will find this plan a low-cost way to provide funds if one of the two dies. It also may be used by husband and wife or father and son to provide funds to pay off special debts or for any of the other purposes for which life insurance may be used.

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An HBA Life Agent would be glad to discuss it further at your convenience.

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4. Articles are accepted for publication only if they are contributed exclusively to this Journal. Ordinarily, contributors will be notified within 60 days if a manuscript is accepted for publication. Every effort will be made to return unused manuscripts

5. The Journal reserves the right to edit all material.

6. Reprints will be supplied to the author at printing cost.

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120 West Fine
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Wednesdays, Weekly, 1 p.m.
Arizona State Hospital, Phoenix, AZ

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CONTACT:
Howard E. Wulsin, M.D.
Arizona State Hospital
2500 E. Van Buren
Phoenix, AZ 85008

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Arizona Medical Center, Tucson, AZ, Rm. 8403

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CONTACT:
Roy N. Killingsworth, M.D.
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Arizona Medical Center
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Last Tues. of Month
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Begin 7/3/74

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CONTACT:
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6037 W. Elm St.
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Every Friday at Noon

Kiva Conference Room
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333 West Thomas Road
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Third Monday of the Month

Kiva Conference Room
Phoenix Memorial Hospital

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CONTACT:

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1201 South 7th Avenue
Phoenix, AZ 85007

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Mondays — 12 Noon

D-5 North Conference Rm., Good Samaritan Hospital, Phoenix, AZ

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Good Samaritan Hospital
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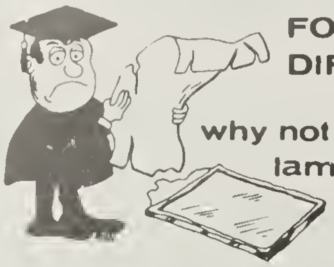

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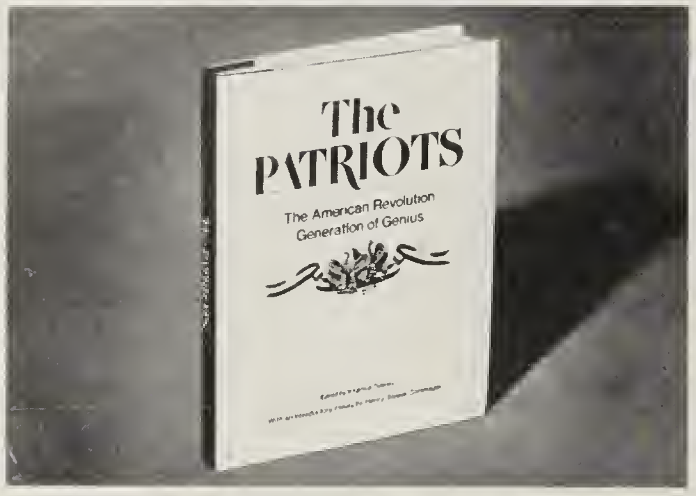

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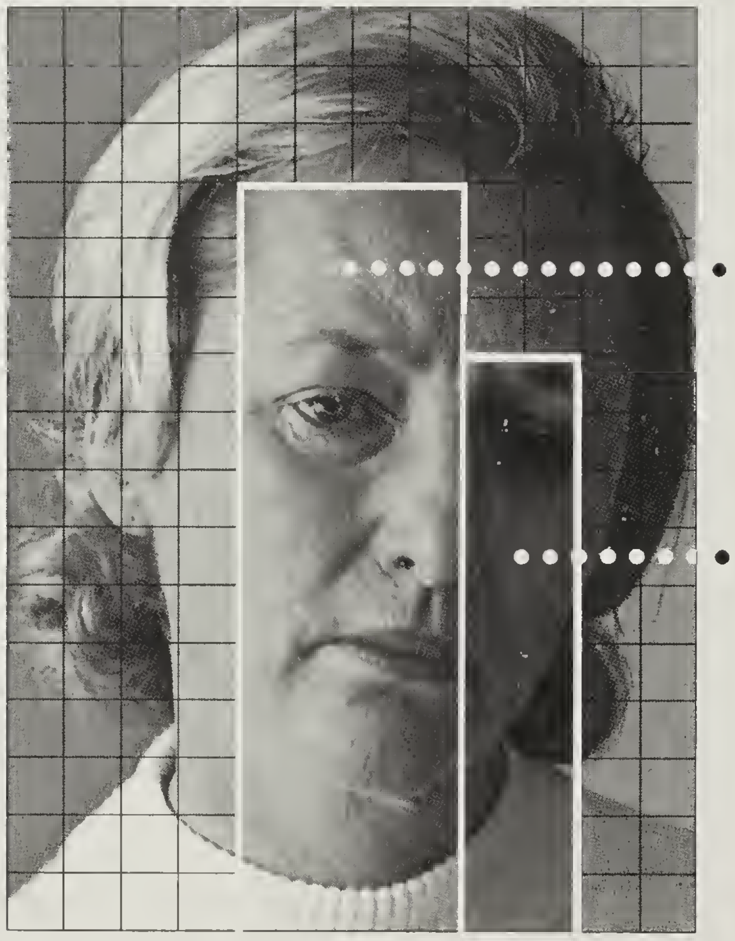
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THERE ARE A LOT OF PEOPLE GETTING BETWEEN YOU AND YOUR PATIENT.

Medicine today is in the spotlight, subjected to all kinds of scrutiny. Your control over patient therapy is being monitored, judged and occasionally abrogated, sometimes by unknown third parties.

The worry is that in the wake of this focus, the relationship between you and your patient will be weakened, without offsetting benefits. Consider three examples:

Drug substitution In most states, pharmacy laws, regulations or professional custom stipulate that your non-generic prescriptions be filled with the precise products you prescribe. But in the last five years, a dozen or more State laws have been changed, permitting the pharmacist in most cases to select a product of the same generic drug to fill any prescription.

Ironically, this dilution of physician control has taken place against a background of growing evidence that purportedly equivalent drug products may be inequivalent, since neither present drug standards nor their enforcement are optimal. In fact, the FDA itself says it has not enforced the same standards for hundreds of "follow-on" products that it had applied to the original NDA approvals. Thus physician control over patient therapy is being eroded with a risk that patients may be exposed to drugs of uncertain quality.

The major advertised claim for substitution is reduced prescription prices for consumers. Yet no documentation of any significant savings has been produced.

MAC Maximum Allowable Cost, MAC for short, is a Federal regulation designed to cut the Government's drug bill by setting price ceilings for drugs dispensed to Medicare and Medicaid patients. Unless the prescriber certifies on the prescription that a particular product is medically necessary, the Government intends to pay only for the cost of the lowest-priced, purportedly-equivalent, generally-available product. The effect of the program may be that elderly and indigent patients will be restricted to products which someone in Washington believes are priced right. Practicing doctors will have little to say about administration of the program, since Government will have absolute authority to make its choices stick.

The drug lag The future of drug and device research depends upon a scientific and regulatory environment that encourages therapeutic innovations. The American pharmaceutical industry annually is spending more than $1 billion of its own funds and evaluating more than 1,200 investigational compounds in clinical research. Disease targets include cancer, atherosclerosis, viruses and central nervous system disorders, among others. But there is a major barrier to the flow of new drugs to your patients: The cost of the research is more than ten times what it was, per product, in 1962; and whereas governmental clearance of new drug applications took six months then, it commonly consumes two years now.

The FDA needs adequate time, of course, to consider data. But it is equally clear that the present approval process contributes to needless delay of needed therapy. That's why the increased efficiency of the drug approval process is vital to all our futures.

If these issues concern you, we suggest that you make your voice heard—among your colleagues and your representatives in State legislatures and in Washington.

It could make a difference in your practice tomorrow.

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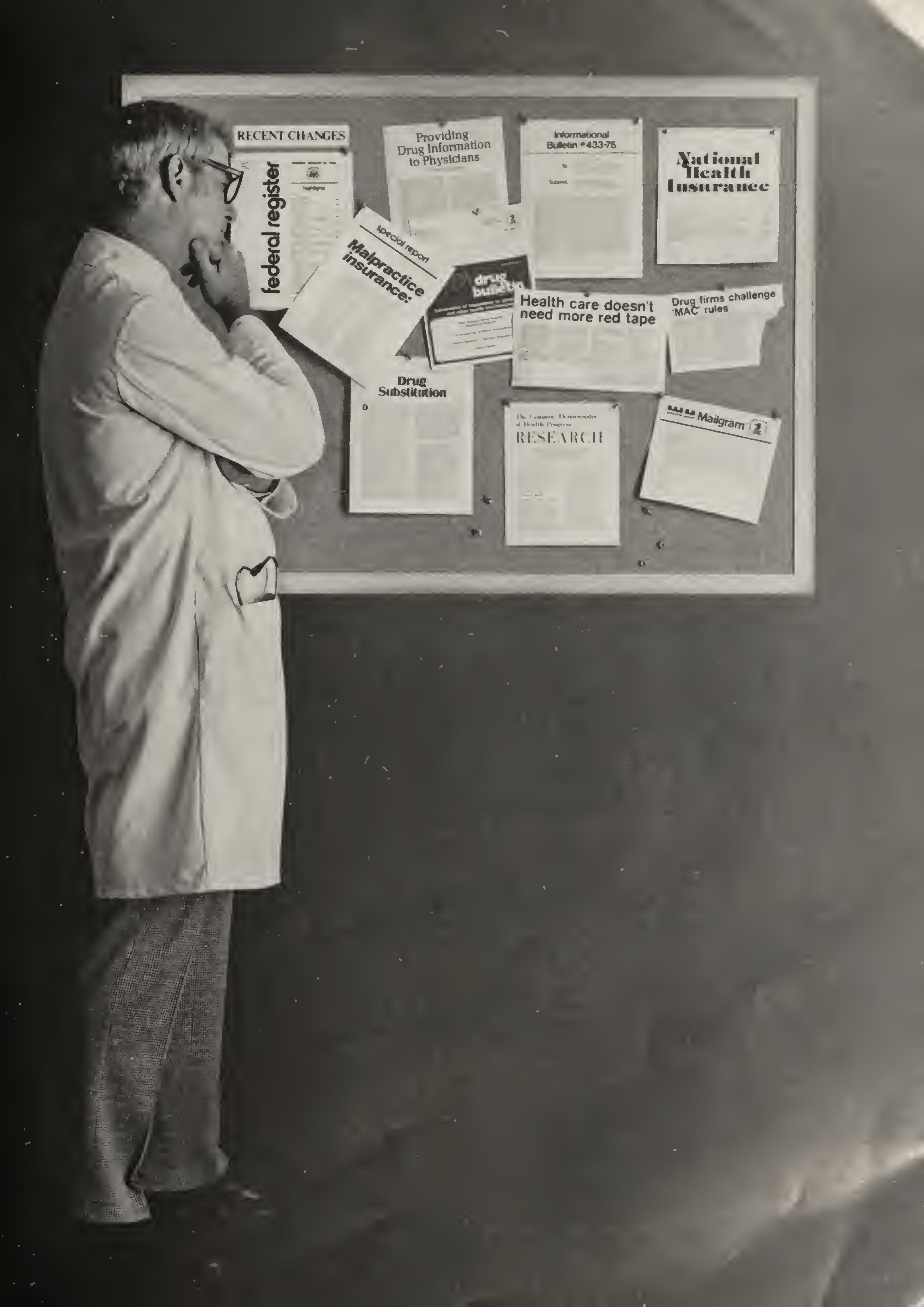
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Original Articles

THE INFECTED CAT BITE

TYPE CASE OF PASTEURELLA MULTOCIDA INFECTION

DOUGLAS LINDSEY, M.D., DR. P.H.
FREDERICK J. HIRSCH, M.D.

This article is an interesting alert to a problem that is apparently more frequent than we realize. Being made aware of it will lead to the diagnosis being made more frequently. However, it will affect the morbidity of mortality very little.

Pasteurella multocida is a relatively obscure, but not infrequent, cause of infection following the bite of a cat (or dog). Suspect *P. multocida* whenever infection is clinically obvious in less than 24 hours following the organism is sensitive to penicillin, and infections respond well to penicillin V in customary oral dosage.

Pasteurella multocida is the currently accepted terminology for an organism formerly classified as several different species: *Pasteurella oviseptica, P. suiseptica, boviseptica,* and others. As evidenced by the prior designations it is capable of causing a variety of animal diseases, but nevertheless is properly regarded as a normal inhabitant of the oropharynx in the same species. P. multocida is of major economic importance as the etiologic agent of a lethal hemorrhagic septicemia in poultry.

Pasteurella multocida is a small, pleomorphic, poorly-staining, Gram negative rod, which may excape notice in the routine processing of clinical culture specimens. It grows readily enough, in air, on blood agar; but it causes no hemolysis, the colonies are small and faint gray, and they tend to get lost in a crowd.

P. multocida does not grow in the presence of bile; consequently it drops out of further consideration when media such as MacConkey's agar

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are utilized as the primary point of separation and identification of aerobic Gram negative rods.

In spite of its shy and seclusive nature you can find *Pasteurella multocida* if you make a reasonable effort to look for it. The microbiology laboratory of University Hospital found it, isolated it, and identified it in 21 patients during the period 1 July 1973 to 31 December 1975. Although some authors[1] have noted a suggestion of seasonal incidence others have not[2]; we are hard put to make anything of the fact that nearly two thirds of our cases—13 in all—were seen in four scattered months of the 30 month period: August 1973, December 1974, March 1975, and December 1975.

The first of the 21 isolations was from an infected cat bite which we treated in the Emergency Room. We missed the clinical diagnosis; it was the first case of human *Pasteurella* infection we had ever seen. The missed diagnosis increased our curiosity and suspicion, and we correctly identified the next case—12 days later. Since then interest has spread among the laboratory and emergency staffs to determine whether or not *P. multocida* is a common or clinically identifiable cause of animal related infections.

Of the 21 isolations, all but three were made from patients treated in the Emergency Room. One was a referral for direct admission to Pediatrics; one was a direct referral to Surgery; and one was an admission from the Oncology/Hematology Clinic to the medical wards.

After missing the first case, we reviewed the findings, refreshed our memory of the literature[3,4,5], *and formulated some tentative clues for the diagnosis of Pasteurella* infection:

1. Animal related wound
2. Rapid onset of infection
3. Signs not typical of either "cellulitis" or "abscess"
4. No organisms found on smear

This formulation of clues remained unchanged until initiation of case review in late December 1975. Informally, and with no great emphasis, it has been passed on to successive rotations of interns and residents as the occasion indicated, usually with elaboration comparable to the following:

Animal related. Typically an infection following cat bite or scratch, but not rare in dog bites. No direct animal contact required; suspect *Pasteurella* in an infected rope burn or fence wire scratch.

Rapid onset. Clinical signs of infection often evident within a few hours after injury. Signs local, and not alarming in intensity; the patient may not be confident on establishing time of onset. In strange contrast to the rapid appearance of local signs of infection, the rarely occurring deep focal or systemic infections may become manifest only after weeks or months of apparent dormancy.[6] Once established, local infection neither progresses inexorably nor necessarily resolves completely; low grade infection may persist for days or weeks.

Signs not typical. It is hard to describe precisely what a classical *P. multocida* infection looks like. The best summary we can give is that it is some sort of a hybrid between "strep" and "staph". The first manifestation is often a narrow, sharply defined erythematous border surrounding some, but not all, of the punctures or scratches. This border differs from early streptococcal cellulitis by a magenta hue, and the absence of a palpable margin. As the erythema spreads it loses the sharp demarcation of color. Swelling may be extensive, noticeable by increase in circumference of a member, shiny skin, or soft edema, but the margin of the swelling shades quite imperceptibly into normal tissue. Lymphangitis and regional adenopathy are not common. At the site of an individual puncture there may be a 1 mm vesicle of thin pus, or a drop of pus of equivalent volume may be expressed from somewhere along a scabbed scratch. Around such sites there may be a more brawny edema, but there is no fluctuation or induration of such degree as to suggest an abscess.

Nothing in the pus. Medical students often seem surprised at the relative scarcity and atypical appearance of organisms on Gram smears of common purulent infections. *P. multocida* is the extreme case. Granulocytes are there in profusion, and well preserved, but a quick scan of the slide shows no Gram positive cocci, and only a patient and painstaking search of many fields—perhaps 30 or more—will turn up one or two faintly stained cocco-bacillary forms.

How well have these clues served us? Not too well.

In only six of the 21 cases was a presumptive diagnosis of "*Pasteurella multocida* infection" made at the time the specimen was submitted for culture; five of these cases were seen by the regular Emergency Room attending staff (the sixth was the case admitted directly to Pediatrics). Findings recorded in the chart indicate that 12 more of the 21 cases could have been diagnosed as *P. multocida* infection by applying the criterion of meeting three out of the four diagnostic clues. (Several of these "recognizable" cases were indeed recognized by attending staff in the course of daily chart audit, but we have no record or tally to establish this.)

The total of "recognized"[6] plus "recognizable"[12] cases would account for more than 80% of the isolations. This percentage is not bad, but we did see half of the 12 "recognizable" cases and failed to make the diagnosis. Worse, the six recognized cases are more than offset by seven cases which were outright false positives: the culture slip was annotated "suspect *P. multocida*", but the organism did not grow out. In summary:

6 recognized, false positive 7
12 recognizable, unsuspected 3

At best then, with all the advantages of hindsight, the four clues appear to be about 60% effective. This is sufficient, we feel, to warrant pointing a firm finger of suspicion, but not good enough to justify a change in therapy from what otherwise would appear to be indicated.

Evaluation of each of the four clues individually gives support to the conclusion that it is possible to simplify the formulation without seriously degrading its clinical utility.

Every one of the 21 confirmed *Pasteurella* infections was the result of direct trauma—bite or scratch—from a cat or dog. Bites outnumbered scratches 3:1; cats outnumbered dogs 2:1—a figure more remarkable in view of the fact that the running ratio of dog bites: cat bites in our E.R. population is 5:1. It would appear that Clue Number 1 could be readily changed from "animal wound" to "bite or scratch of a cat".

If "rapid" onset of infection is defined as "less than 24 hours" the clue retains considerable predictive value. Time of onset was recorded in only 17 cases. The 12 cases with onset less than 24 hours include seven cat bites, and four cat scratches; the five cases with onset greater than 24 hours include four dog bites and one cat scratch.

The authors are not consistent between themselves on evaluation of the clinical signs in an individual case. Overall, we agree that about two-thirds of patients who have a *P. multocida* infection *look* like they do, but we may disagree as to which group a given patient falls in. In consequence we feel it premature to propose that there is indeed a characteristic appearance of *Pasteurella* wound infection until we have developed criteria which are more objective. The best evidence we have that the infection differs in appearance from infections produced by streptococci and staphylococci is that in no instance did a culture positive for *P. multocida* also grow out either beta-hemolytic streptocci or *S. aureus*.

Smears proved to be of little predictive value. Only nine smears were made in the 21 confirmed cases. "No organisms found on smear" had to be denigrated to uncertain debate on the significance of an "Occasional Gram positive cocci" or a "Few Gram negative rods."

It thus appears that a useful clue to the clinical assessment of possible acute *P. multocida* infection can be formulated quite simply as:

Clinically evident infection,
occuring within 24 hours
of a bite or scratch of a cat.

Application of this modified formulation to the records of the cases discussed above indicates that it would have picked up 11 of the 14 *Pasteurella* infections from cat bites (or scratches), left three unsuspected, and cut the false positives from seven to four.

Other than the value of satisfaction of the ego of the clinician (considerable) the successful pre-culture diagnosis of *Pasteurella multocida* infection has only one practical application: economical treatment. *Pasteurella multocida* is sensitive to penicillin. It is also sensitive to almost everything else. If you are already treating the patient with dicloxacillin or cephalexin then stay with it; the prescription is already bought. If you have not yet initiated therapy, and if there is either no exudate to smear or there are Gram positive cocci on smear, we think it wiser to start with dicloxacillin. If the smear is free of Gram positive cocci, we start with oral phenoxymethyl penicillin (penicillin V). It saves the patient money. And that's no bad thing nowadays.

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A PROPOSAL

SOME NEW, "OLD", TERMS AND A SCORING SYSTEM FOR IRREVERSIBLE COMA

GORDON M. GREENBLATT, M.D.

Advances in resuscitative techniques, life support systems and transplant surgery have all made what was an apparently simple determination of death a highly complicated one. The author proposes a scoring mechanism involving the functions of the cental nervous, cardiovasculature and respiratory systems which may hopefully give us at least a semi-quantitative evaluation of the problem.

"Criteria, any criteria, do not define death. Rather they can allow us to say only that death has occurred. It is detection rather than definition."[1]

"Various abstract terms listed . . . do not contribute to the devising of . . . criteria . . . that a man has died. They are, perhaps, be avoided."[2]

"What dies is the organism as a whole. It is this death, the death of the individual human being, that is important for physicians and for the community, not the "death of organs or cells, which are mere parts."[3]

"The same considerations that apply to abortion would appear to apply, in principle, to decisions at the other end of life."[4]

Perhaps it is foolhardy to propose old terms for new purposes. The problem of clearly communicating the death of an individual to lay persons is one obstacle in permitting all those involved in such an event to agree that death actually has occured.

Our collegues in obstetrics solve this dilema with fairly precise terms when dealing with abortion. "Threatened," "Impending," "Inevitable," "Spontaneous," and "Complete" serve most admirably in communicating the death or potential death of a human fetus to the medical and lay community. These terms are not entirely abstract and can be defined by the dilatation of the cervix, and/or retention of the abortus.

In combining such terms, along with a scoring system (not unlike the obstetrical "Apgar" score), based on acceptable criteria for the detection of death, we as doctors, can hopefully convey to lay persons that not only has the individual actually died, but that the existence of any "life" in body organs, is not a prognosis that the "individual" might again or still be considered alive.

Such a scoring must of necessity be abstract in concept, but detailed in the individual application. Hopefully after continued experience, this will become a useful system.

"The new criteria are meant to be necessary for only that small percentage of cases where there is irreversible coma with permanent brain damage, and where traditional signs of life are obscured because of the intervention of resuscitative machinery."[2]

From: 5800 N. 19th Ave., Suite 204, Phoenix AZ 85015. Submitted for publication 3/76.

As with the above commentary on the Harvard Medical School Criteria, I also exclude any coma caused by hypothermia or central nervous system depressants.

The method consists of an addition of points assigned to various findings in the central nervous, cardiovascular, and respiratory systems which results in the total "score" that corresponds to a descriptive term.

The terms appropriate for this purpose are: Threatened death, inevitable death, and complete death. Score from 0 to 28 determines which term is applicable. "Threatened death" from 4 to, or equal to 6; "Inevitable death" from greater than 6 to or equal to 12; and "Complete death" greater than 12.

Fortunately, my practice of anesthesiology does not provide many patients with irreversible coma. In two instances in the Intensive Care Unit, this system did convey to the patient's families that their relative was indeed clinically dead.

The scoring is purposely weighted toward high scores for "complete death', so that on the scale of 0 to 28, there can be no doubt as to the finality of the situation.

Again this should only be considered a method of communicating to a lay person, in numbers, the already medically determined fact of death.

I. Central Nervous System:	**Points**
(1) Awake and responsive	0
(2) Depressed—reacts to external stimuli with appropriate reflexes—Pupils react to light.	1
(3) Depressed—pupils dilated—respond to light—no response to painful external stimuli and/or pathological reflexes.	2
(4) Pupils bilaterally dilated and fixed—flaccid without responses of any type to any external painful stimuli—absent corneal reflex.	6
(5) Isolectric EEG recorded spontaneously and during auditory and tactile stimulation with multiple recording for 30 seconds using standard number of electrodes with maximum allowable electrode distance—Point of recording at full gain with external artifacts ruled out with right hard electrode. Recorded twice, 24 hours apart.	10
(6) Cerebral oxygen consumption less than 10% normal or angiographic evidence of failure of cerebral circulation.	12

II. Cardiovascular System:	
(1) Normal cardiac electrical activity and self sustaining blood pressure.	0
(2) Normal cardiac electrical activity and blood pressure sustained chemically or by assisted or controlled respiration.	2
(3) Abnormal cardiac electrical activity and blood pressure sustained chemically or by assisted or controlled respiration.	6
(4) Dying heart on EKG and/or no electrical activity with no blood pressure.	8

III. Respiration:	
(1) Spontaneous and adequate.	0
(2) Spontaneous inadequate with normal pattern, may require mechanical assistance.	2
(3) Abnormal pattern of breathing—Inadequate or adequate, may require mechanical assistance.	6
(4) Apnea—requiring controlled assistance and continuing three to five minutes after cessation of mechanical control with room air and prior normal carbon dioxide levels	8

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SPOROTRICHOSIS

IN SOUTHERN ARIZONA

CASE REPORT

JAMES T. SINSKI, PH.D.
OTIS B. MILLER, M.D.
W. DAVID JACOBY, M.D.

Diagnosis of unusual or rare disorders is more apt to be made if the physician keeps an awareness of its features current. This article should make physicians cognizant of the fact that Sportrichosis does occur in Arizona.

SYNOPSIS ABSTRACT

This is the first case of sporotrichosis reported to have been acquired in Arizona. The gross appearance of the primary lesion on the hand, the difficulty to observe the fungus in histologic section, the isolation of the mycelial phase of the organism and subsequent transition to the yeast phase, the pathogenicity of the organism for mice, and the weather conditions during the time of infection are all similar to those already described by others. No predisposing conditions were found in the patient, a 55 year old male wood cutter, who contaminated a cut on his right middle finger with sawdust from a rotted ash tree while working near Pearce, Arizona.

There is no published report of a human case of sporotrichosis acquired in Arizona. Though at least two cases have been seen by dermatologists for diagnosis and treatment in Tucson within the past 10 years, both were residents of the State of Sonora, Mexico, where it is assumed, they acquired their infection.

CASE HISTORY

A 55 year old male woodcutter, who had been in excellent health, gave the history of having cut his right middle finger while chopping a tree in the mountain area (elevation 5184 feet) near Pearce, Arizona. He stated that the wound was contaminated by sawdust from the rotted base of an old ash tree. Following the development of a small pustule on the finger, at a time not precisely stated by the patient, a red streak running up the forearm was noted within a week. This was treated with numerous drug store remedies to no avail.

Two months later in January, 1976, he was seen by two of us (O.B.M. and W.J.D.). At that time he had 8 raised, tender, purplish-red skin tumefactions arranged in linear fashion from the right middle finger up the extensor surface of the wrist and forearm. On the flexor aspect of the right forearm there were 3 similar lesions. These lesions, measuring 1 to 2 cm in diameter, were surfaced with scaling and hemorrhagic crusts. There was a distinct erythematous streak, extending from the antecubital space up the flexor surface of the right arm. The general health of the patient was unaffected.

To confirm the clinical diagnosis of sporotrichosis, punch biopsies were taken for histologic examination and culture. Therapy with aqueous potassium iodide in gradually increasing doses was prescribed and tolerated well. On 12 March 1976 after approximately 2 months of therapy, the lesions were noted to be flatter, less erythematous, and less tender.

LABORATORY:

A Histology

Sections stained with H & E and PAS with diastase predigestion demonstrated a granulomatous reaction with foci of necrosis but failed to reveal the causal organism. Few yeast cells were seen in Gomori methenamine silver stained tissue.

B. Mycology

The tissue was ground in saline and inoculated on Sabouraud's plus cycloheximide and chloramphenicol, and DTM.[1] Both of the media

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Reprint Requests to: Department of Microbiology and Medical Technology, University of Arizona, Tucson Arizona 85721.

supported the growth of the mycelial phase of the organism at room temperature, and it was obtained in pure culture. After the organism was visible for about 5 days, the DTM medium turned red. Grossly and microscopically, the isolated mycelial organism had the morphology of *Sporothrix schenckii*.[2] Because of low relative humidity in Tucson, some difficulty was encountered in converting the mycelial phase to the yeast phase. Yeast phase was not obtained upon culture of the ground tissue because the surface of the media did not remain moist. Conversion from mycelial phase to yeast phase was achieved on brain-heart-infusion agar slant when grown at 37°C in small confined incubator that had little air circulation. Complete conversion to yeast phase was achieved with the second serial transfer. Yeast phase was found to be pathogenic for mice using intratesticular route of injection. Repeated attempts to isolate the organism from pieces of wood and forest litter supplied by the patient in February 1976 were uncuccessful. This was a period of 3 months after the initial infection.

COMMENT:

Sporotrichosis can simulate granulomatous lesions of almost any other origin. An ulcer may be gummatous or may simulate an epithelioma. The classical form is a chanere-form lesion with satellites which may occur with any of the deep mycoses. Plaques may suggest tuberculoid leprosy or sarcoidosis. Vascular lesions resembling polyarteritis nodosa may be found.[3] The causative organism *S. schenckii*, may remain localized in the subcutaneous tissue, may spread locally in the subcutaneous lymphatics, or may rarely, be widely disseminated in the blood stream. The immunological response of the host undoubtedly determines the form which the infection assumes.[3] Roberts[4] reports approximately 50% of the total number of cases of sporotrichosis to occur in farmers, laborers, gardeners, and horticulturists. The causal organism is known to grow on a variety of live and dead vegetable materials and wood splinters have been specifically reported to be one of the sources of infection.

The average temperature reported by a number of investigators during which most of the infections with *S. schenckii* occur was 15°C or approximately 59°F.[2] The average temperature for the month of October 1975 as reported for Pearce, Arizona, was 63.1°F.[5] High relative humidity does not appear essential for growth of the organism in nature. High incidence of the infection has been reported during periods of both high and low relative humidity. The fungus appears to be resistant to drying.[2] The relative humidity for Pearce, Arizona is not recorded but the total precipitation for the month was 0.25 inches.[5]

As far as therapy is concerned, the drug of choice for cutaneous sporotrichosis is potassium iodide. In the systemic type, where bones,joints, and any organ may be involved, intravenous amphotericin B is necessary.

ACKNOWLEDGMENTS

The chloramphenicol was graciously supplied by Parke, Davis and Co., Detroit, Michigan.

WORK SPONSORED

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GENERIC AND TRADE NAMES OF DRUGS

Cycloheximide — Actidione
Chloramphenicol — Chloromycetin

INDEXING TERMS

Sprortrichosis
Sporothrix schenckii

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ERRATA

OBSERVATIONS OF VIRUS-LIKE PARTICLES IN A CARCINOMA OF THE PENIS, June issue, Page 150. Reprint requests should be sent to Murray S. Feldstein, M.D., 900 N. San Francisco, Flagstaff, AZ 86001

SUMMER DOLDRUMS

John W. Kennedy, M. D.

The Roentgen Revolution has a recent spin-off, which has aroused the medical politic, even more than the eminent threat of National Health Insurance.

It is not our province to suggest which physicians should man these esoteric monsters, or where the apparatus should be placed i.e., hospitals or private offices or clinics. But, there are economic problems and the current literature reflects these facets.

Most of the preliminary reports indicate the fantastic diagnostic uses. Invasive and expensive x-ray procedures such as cerebral angiography, cerebral pneumoencephalography and ventriculography, all accompanied by a low but significant mortality and morbidity, these procedures may become far less necessary in skull and brain diagnosis.

The economic impact has aroused the health planners at federal, state and local levels.

A report to the Senate subcommittee on health requested from the office of Technical Assessment in Washington brings this into focus. These are excerpts from the report, Dr. H. David Bauta, specialist in preventive medicine, principle author:

"Hospitals and other facilities commonly charge $220.00 for each patient undergoing a CAT scan, the Cleveland Cinic has been able to reduce the charge to $100.00. This testimony and other evidence on the race to acquire the expensive new devise is likely to impel Congress to consider ways of controlling the spread of scanners."

"Sixteen states have tried to assert controls over these installations." And in many instances this applies to in and out of hospital installations. The Central Arizona Health Systems Agency— part of the long arm of HEW has a committee mulling the problem.

CAT Fever—as the New England Journal of Medicine denotes it as it describes the "epidemic." Although the exact number of units existing in the U.S. is uncertain, the capital costs for the. . .units have been estimated, at todays prices as 1.2 billion dollars over the next decade."

Then there is as described, the other experiences we have seen in examples of advanced radiologic and surgical technics. Mass screening for carcinoma of the stomach and bowel cancer, even mass screening for pulmonary tuberculosis is no longer worth the effort. "Others have expressed grave reservations regarding the ultimate effect of coronary artery bypass grafts on morbitiy and morality."

The New York Times puts the problem in pragmatic terms, it "may revolutionize the diagnosis and treatment of many internal ills or merely add to the already staggering costs of health care."

Then there is the "conflict of interest facet. How many hospitals physicians and clinics taking on the investment opportunity which may accompany marketing of some of the devices, now find themselves saddled with a monthly mortgage payment that might create a conflict of interest which could influence useage." M. T. Rubkin, *Boston Science.*

And finally this "with a high rate of use, experts say x-ray scanners can generate $2,000 to $3,000 of revenue a day and pay for themselves in a year or less. So far the health profession has encountered little price resistance from health insurers that pay the bills for scanning." *Wall Street Journal*

Advance in medical technology are now under economic scrutiny, and like the "moon shots" can we really afford them.

> *"Some of the most responsible doctors will always be in the hands of financial fakers, and some of the most responsible business men will always be in the hands of medical fakers."*
>
> Robert Morris

AM

Editor's Page

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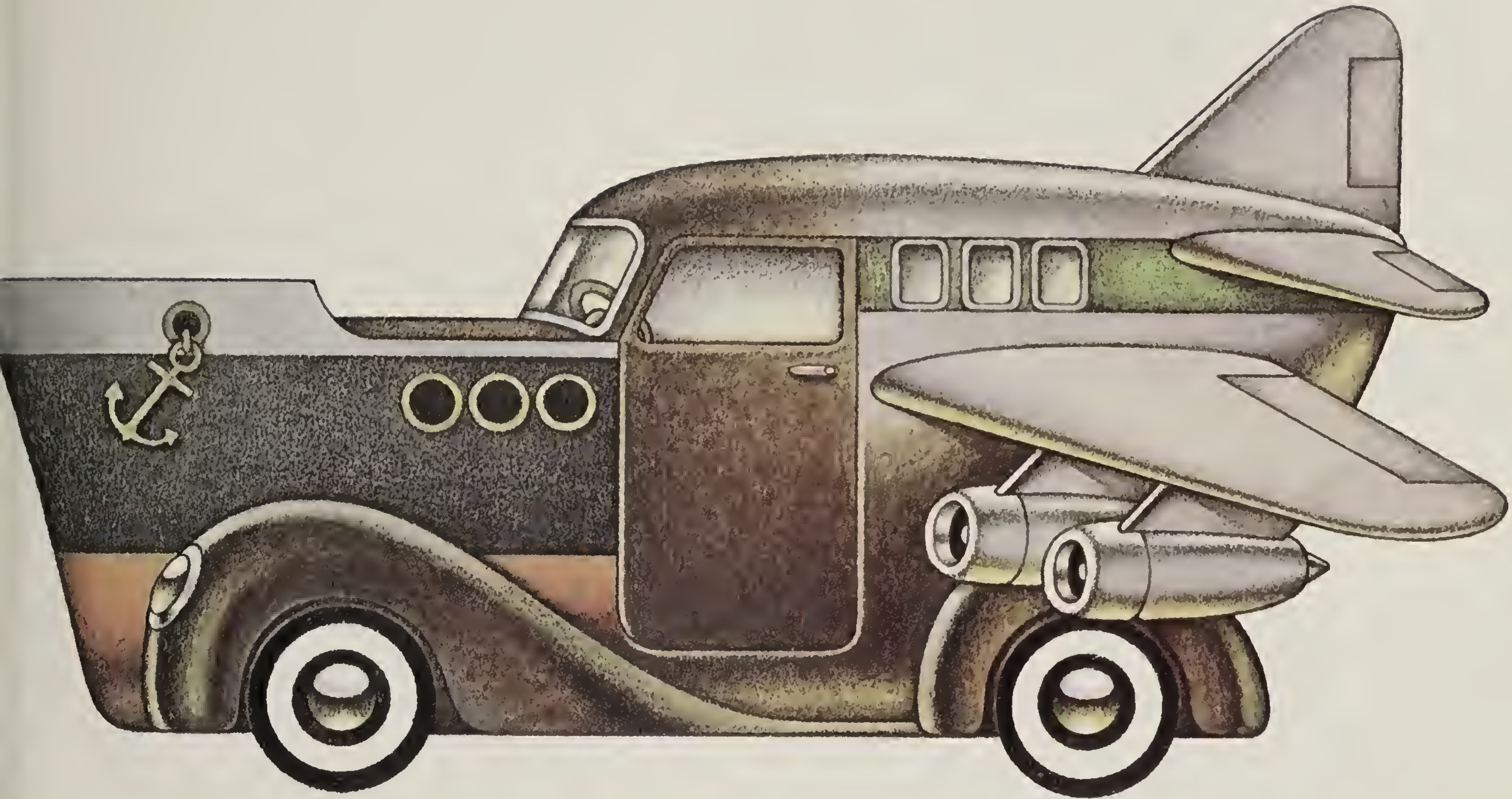

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Up to 24 hours of effective control with a single dose...in nausea, vomiting and dizziness associated with motion sickness.

Dosage: 25 to 50 mg. 1 hour before travel.
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BRIEF SUMMARY OF PRESCRIBING INFORMATION

CONTRAINDICATIONS. Administration of Antivert during pregnancy or to women who may become pregnant is contraindicated in view of the teratogenic effect of the drug in rats.

The administration of meclizine to pregnant rats during the 12-15 day of gestation has produced cleft palate in the offspring. Limited studies using doses of over 100 mg./kg./day in rabbits and 10 mg./kg./day in pigs and monkeys did not show cleft palate. Congeners of meclizine have caused cleft palate in species other than the rat.

Meclizine HCl is contraindicated in individuals who have shown a previous hypersensitivity to it.

WARNINGS. Since drowsiness may, on occasion, occur with use of this drug, patients should be warned of this possibility and cautioned against driving a car or operating dangerous machinery.

Usage in Children: Clinical studies establishing safety and effectiveness in children have not been done; therefore, usage is not recommended in the pediatric age group.

Usage in Pregnancy: See "Contraindications."

ADVERSE REACTIONS. Drowsiness, dry mouth and, on rare occasions, blurred vision have been reported.

ROERIG Pfizer
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Antivert®/25 Chewable Tablets
(meclizine HCl) 25 mg.
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DYAZIDE® MAKES SENSE FOR LONG-TERM CONTROL OF HYPERTENSION*

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Each capsule contains 50 mg. of Dyrenium® (triamterene, SK&F Co.) and 25 mg. of hydrochlorothiazide.

Before prescribing, see complete prescribing information in SK&F literature or *PDR*. The following is a brief summary.

* **Warning**
This fixed combination drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual patient. If the fixed combination represents the dosage so determined, its use may be more convenient in patient management. The treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** *Edema.* That associated with congestive heart failure, cirrhosis of the liver, the nephrotic syndrome; steroid-induced and idiopathic edema; edema resistant to other diuretic therapy. *Mild to moderate hypertension:* Usefulness of the triamterene component is limited to its potassium-sparing effect.

Contraindications: Pre-existing elevated serum potassium. Hypersensitivity to either component. Continued use in progressive renal or hepatic dysfunction or developing hyperkalemia.

Warnings: Do not use dietary potassium supplements or potassium salts unless hypokalemia develops or dietary potassium intake is markedly impaired. Enteric-coated potassium salts may cause small bowel stenosis with or without ulceration. Hyperkalemia (>5.4 mEq/L) has been reported in 4% of patients under 60 years, in 12% of patients over 60 years, and in less than 8% of patients overall. Rarely, cases have been associated with cardiac irregularities. Accordingly, **check serum potassium during therapy, particularly in patients with suspected or confirmed renal insufficiency (e.g., elderly or diabetics). If hyperkalemia develops, substitute a thiazide alone. If spironolactone is used concomitantly with 'Dyazide', check serum potassium frequently**—both can cause potassium retention and sometimes hyperkalemia. Two deaths have been reported in patients on such combined therapy (in one, recommended dosage was exceeded; in the other, serum electrolytes were not properly monitored). Observe patients on 'Dyazide' regularly for possible blood dyscrasias, liver damage or other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving Dyrenium (triamterene, SK&F). Rarely, leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with the thiazides. Watch for signs of impending coma in acutely ill cirrhotics. Thiazides are reported to cross the placental barrier and appear in breast milk. This may result in fetal or neonatal hyperbilirubinemia, thrombocytopenia, altered carbohydrate metabolism and possibly other adverse reactions that have occurred in the adult. When used during pregnancy or in women who might bear children, weigh potential benefits against possible hazards to fetus.

Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis; rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting (may indicate electrolyte imbalance), diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 capsules; in Single Unit Packages of 100 (intended for institutional use only).

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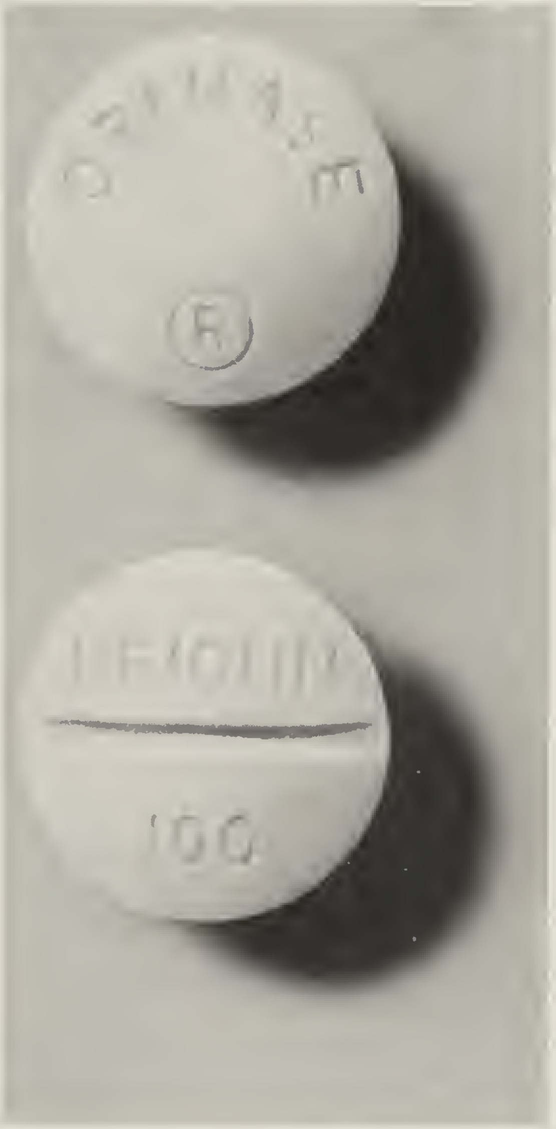

This new design will help pharmacists, physicians, nurses, and patients identify Orinase by name and manufacturer. The number on the tablet is for identification and is not related to tablet strength.

You may wish to advise your patients that this change is taking place.

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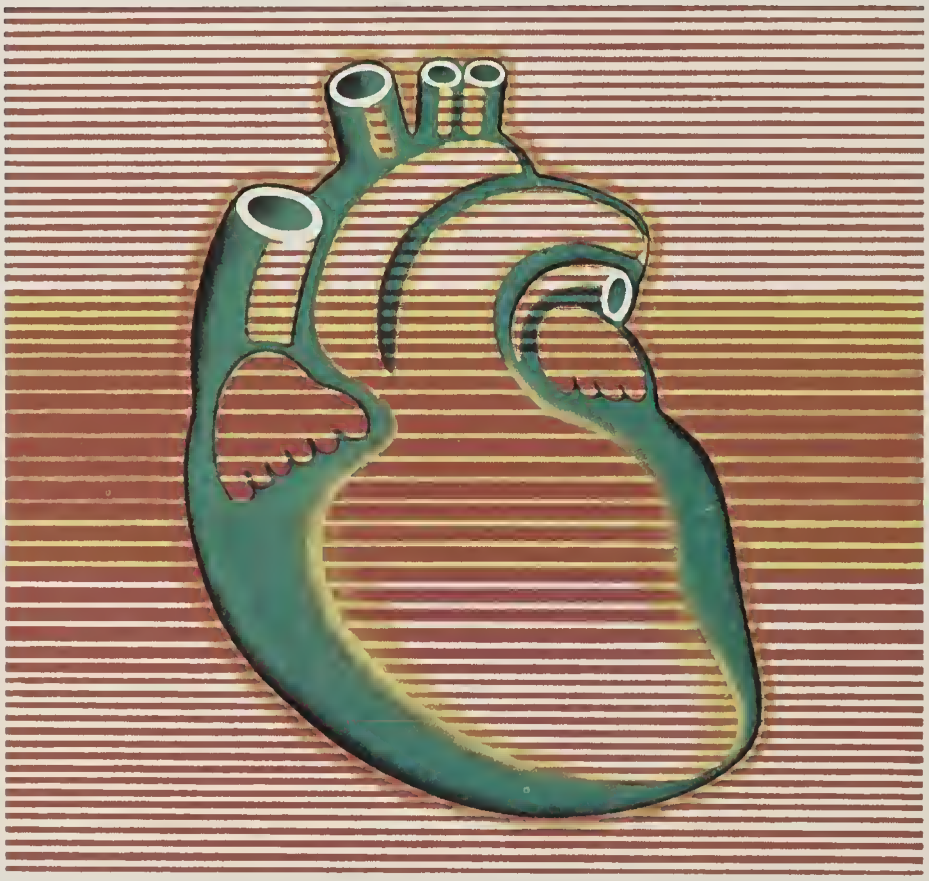
WHEN ANXIETY INTERFERES.

The cardiac patient and anxiety.



"The [cardiac] patient is anxious about minor symptoms, about the implications of his diagnosis, and about real or imagined limitations of function."*

The worst is over. The cardiac patient is out of the acute stage, out of the hospital, and well on his way to recovery. How quickly he comes back to near normal functioning may depend on his psychological as well as his physical rehabilitation.

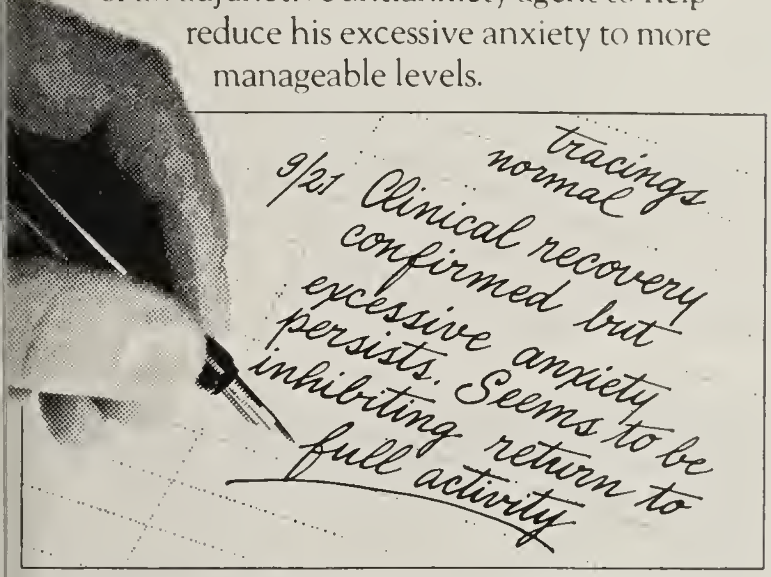
Clinical anxiety, for example, may be one reason for prolonged recuperation following cardiac healing. Yet anxiety can sometimes be beneficial in facilitating patient compliance.

The patient who is realistically concerned about resuming his precoronary functioning may be highly motivated to adhere to his rehabilitation regimen. However, the cardiac patient with *excessive* or unresolved anxiety may be so fearful of future heart failure that he refrains from your prescribed regimen. These excessively anxious cardiac patients eventually present the same clinical characteristics as patients deconditioned by bed rest.

Excessive anxiety can interfere with patient management

When excessive anxiety diminishes your

patient's ability to participate fully in his rehabilitation program, your counseling and reassurance are often sufficient. But when his anxiety is so great that it actually interferes with his ability to listen and respond, you may want to consider the addition of an adjunctive antianxiety agent to help reduce his excessive anxiety to more manageable levels.


tracings normal
9/21 Clinical recovery confirmed but excessive anxiety persists. Seems to be inhibiting return to full activity

Librium® (chlordiazepoxide HCl) an effective adjunct to your reassurance and counseling

Safety: Librium has a highly favorable benefits-to-risk ratio and a wide margin of safety. Because of its low incidence of side effects, it is regarded as one of the safest antianxiety agents available. And Librium does not adversely affect the cardiovascular system. See complete product information for warnings, precautions and adverse reactions.

Performance: Hundreds of clinical trials, thousands of published papers, and millions of patients comprise the record of performance for Librium.

Concomitant use: Of special significance in treating the cardiac patient already taking multiple agents is the fact that Librium is used concomitantly with most primary medications, such as cardiac glycosides, diuretics and antihypertensives.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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THE ANXIETY-SPECIFIC

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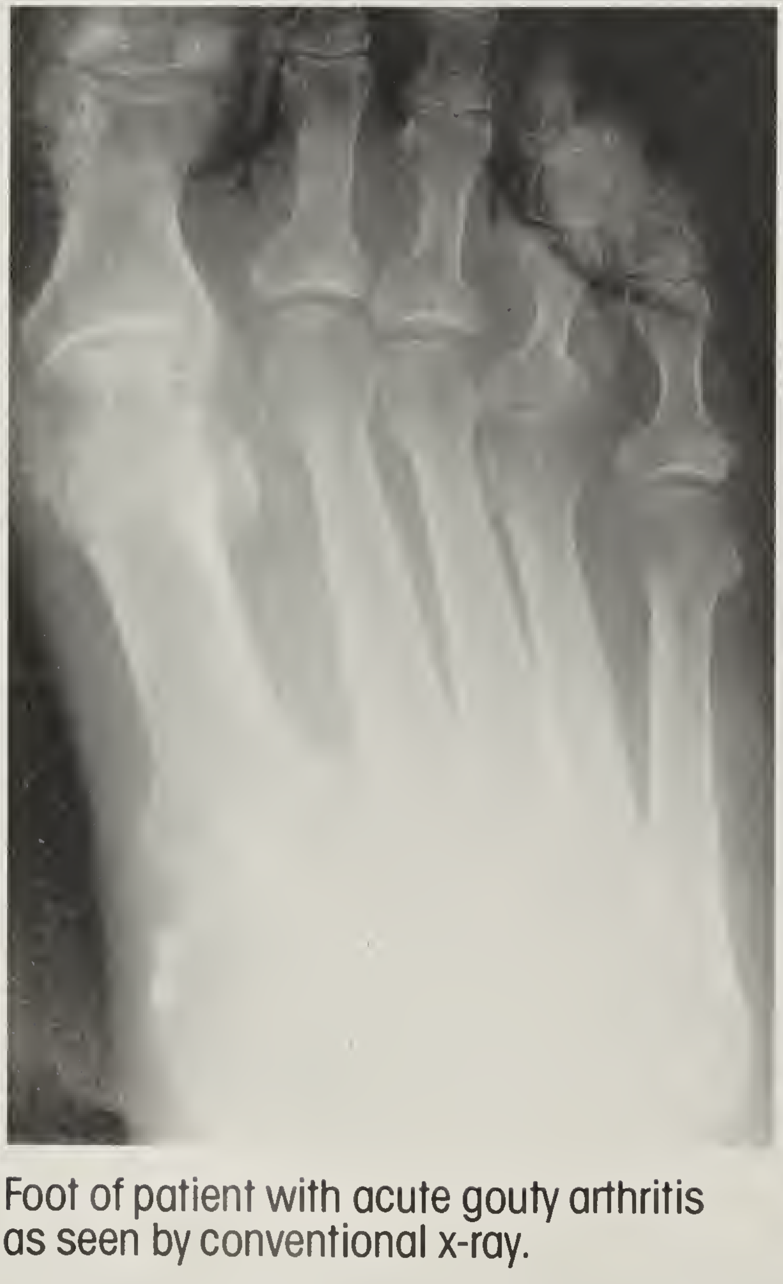
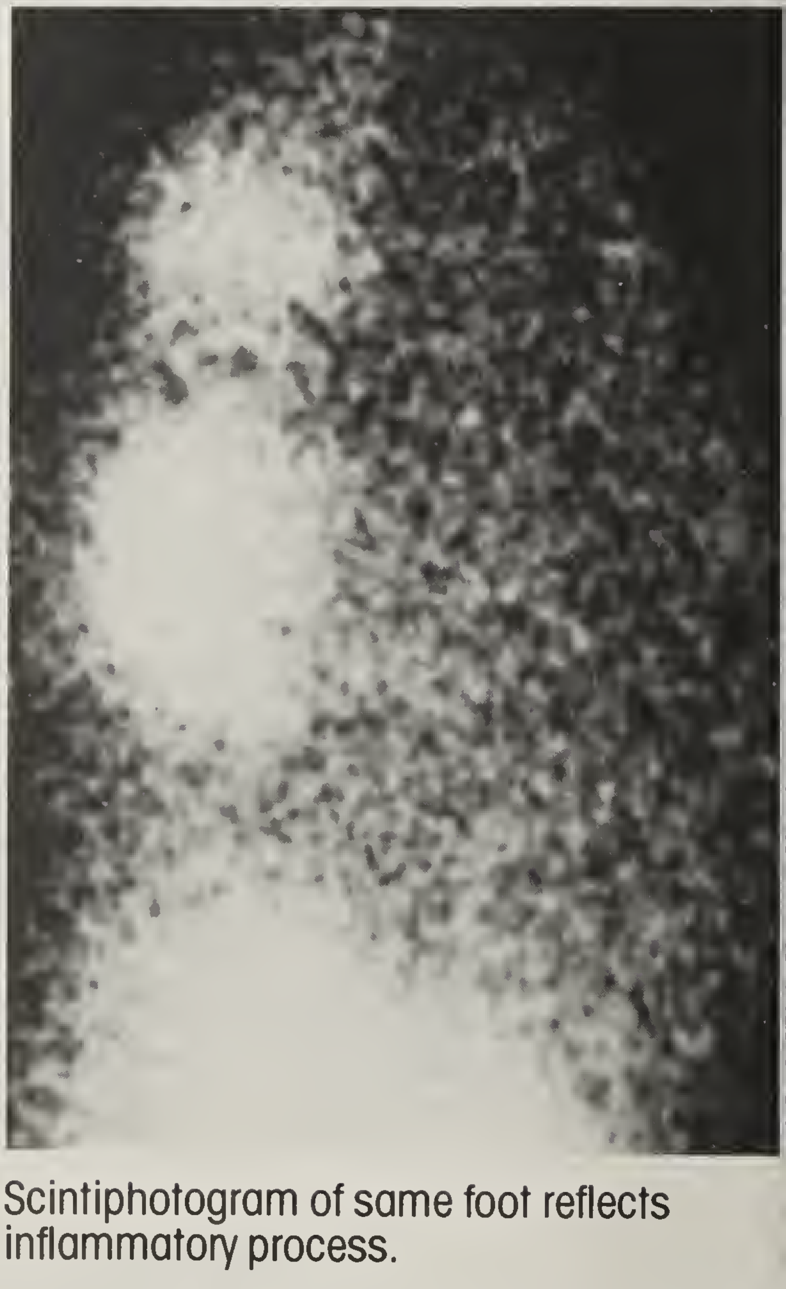
*Zohman BL. *Geriatrics* 28:110-119, Feb 1973

VISUAL FOCUS
ON
ACUTE GOUTY ARTHRITIS



Foot of patient with acute gouty arthritis as seen by conventional x-ray.



Scintiphotogram of same foot reflects inflammatory process.

The scintiphotograph on the right shows increased uptake of radiotechnetium polyphosphate in the metatarsophalangeal joint and the proximal interphalangeal joint of the great toe of a patient with acute gouty arthritis. This increased uptake probably results from increased vascularity in the affected areas.

For a more detailed description of scintiphotography, see "addendum" at right.

THERAPEUTIC FOCUS ON

CAPSULES, 25 mg and 50 mg

INDOCIN®
INDOMETHACIN | MSD)

helps relieve pain and other symptoms of inflammation in acute gouty arthritis in selected patients

INDOCIN is a potent drug with anti-inflammatory, antipyretic, and analgesic properties. It should not be used in conditions other than those recommended. Although INDOCIN does not alter the progressive course of the underlying disease, in selected patients with acute gouty arthritis it has been found highly effective in relieving pain and in reducing fever, swelling, and tenderness.

MSD
MERCK
SHARP&
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For a brief summary of prescribing information, please see following page.

addendum

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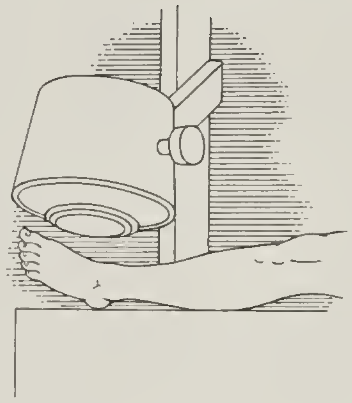
Facts about Scintiphotography



In recent years a variety of radiopharmaceuticals have been employed to aid in the diagnosis of bone and joint disorders. The joint-imaging technique consists of injecting technetium polyphosphate intravenously, and imaging is performed with the scintillation camera two hours after the administration of the radionuclide. In general, for joint surveying, the shoulders, elbows, hands, wrists, knees, ankles, feet, and vertebral column are mapped. The entire scanning process takes approximately one hour. The criterion for a positive image is a higher concentration of radioactivity in a joint region than in adjacent nonarticular bone. In effect, each patient serves as his own control.

INDOCIN®
(INDOMETHACIN | MSD)

helps relieve pain
and other symptoms
of inflammation
in acute
gouty arthritis
in selected patients

IMPORTANT NOTE: INDOCIN (Indomethacin, MSD) cannot be considered a simple analgesic and should not be used in conditions other than those recommended. The drug should not be prescribed for children because safe conditions for use have not been established.

Because of the high potency of the drug and the variability of its potential to cause adverse reactions, the following are strongly recommended: 1) the lowest possible effective dose for the individual patient should be prescribed. Increased dosage tends to increase adverse effects, particularly in doses over 150-200 mg per day, without corresponding clinical benefits; 2) careful instructions to, and observations of, the individual patient are essential to the prevention of serious and irreversible, including fatal, adverse reactions, especially in the aging patient.

Contraindications: Children 14 years of age and under; pregnant women and nursing mothers; active gastrointestinal lesions or history of recurrent gastrointestinal lesions; allergy to aspirin or indomethacin.

Warnings: *Gastrointestinal Effects:* Because of the occurrence and, at times, severity of gastrointestinal reactions, be continuously alert for any sign or symptom signaling a possible gastrointestinal reaction. The risks of continuing therapy with INDOCIN in the face of such symptoms must be weighed against the possible benefits to the individual patient. Gastrointestinal effects may be reduced by giving the drug immediately after meals, with food, or with antacids. Use greater care in aging patients.

Ocular Effects: Corneal deposits and retinal disturbances, including those of the macula, have been observed in some patients on prolonged therapy. Discontinue therapy if such changes are observed. Ophthalmologic examination at periodic intervals is desirable in patients on prolonged therapy.

Central Nervous System Effects: INDOCIN may aggravate psychiatric disturbances, epilepsy, and parkinsonism, and should be used with considerable caution in patients with these conditions. If severe CNS adverse reactions develop, discontinue the drug.

Precautions: Blurred vision may be a significant symptom that warrants a thorough ophthalmologic examination. Patients should be cautioned about engaging in activities requiring mental alertness and motor coordination, as driving a car. Headache which persists despite dosage reduction requires complete cessation of the drug. May mask the usual signs and symptoms of infection; therefore, the physician must be continually on the alert for this and should use the drug with extra care in the presence of existing controlled infection. After the acute phase of the disease is under control, an attempt to reduce the daily dose should be made repeatedly until the patient is off entirely.

Drug Interactions: Although INDOCIN has not influenced the hypoprothrombinemia produced by anticoagulants, patients on anticoagulant therapy should be observed closely for alterations in prothrombin time. In patients receiving probenecid, plasma levels of indomethacin are likely to be increased and a lower total daily dose of INDOCIN may produce a therapeutic effect; increases in the dose of INDOCIN should be made cautiously and in small increments.

Adverse Reactions: *Gastrointestinal Reactions:* Single or multiple ulcerations of the esophagus, stomach, duodenum, or small intestine, including perforation and hemorrhage, with fatalities in some instances; rarely, intestinal ulceration has been associated with stenosis and obstruction; gastrointestinal bleeding without obvious ulcer formation; perforation of preexisting sigmoid lesions (diverticulum, carcinoma, etc.); rarely, increased abdominal pain in ulcerative colitis patients or development of ulcerative colitis and regional ileitis; gastritis may persist after the cessation of the drug; nausea, vomiting, anorexia, epigastric distress, abdominal pain, and diarrhea.

Eye Reactions: Corneal deposits and retinal disturbances, including those of the macula, have been observed on prolonged therapy; blurring of vision.

Hepatic Reactions: Rarely, toxic hepatitis and jaundice, including some fatal cases.

Hematologic Reactions: Aplastic anemia, hemolytic anemia, bone marrow depression, agranulocytosis, leukopenia, and thrombocytopenic purpura may occur rarely. Since some patients manifest anemia secondary to obvious or occult gastrointestinal bleeding, appropriate blood determinations are recommended.

Hypersensitivity Reactions: Acute respiratory distress, a rapid fall in blood pressure resembling a shock-like state, angioedema, dyspnea, asthma, angiitis, pruritus, urticaria, skin rashes, purpura.

Ear Reactions: Hearing disturbances—deafness reported rarely; tinnitus.

Central Nervous System Reactions: Psychic disturbances including psychotic episodes, depersonalization, depression, and mental confusion; coma; convulsions; peripheral neuropathy; drowsiness; lightheadedness; dizziness; syncope; headache.

Cardiovascular-Renal Reactions: Edema, elevation of blood pressure, hematuria.

Dermatologic Reactions: Loss of hair, erythema nodosum.

Miscellaneous: Rarely, vaginal bleeding, hyperglycemia, glycosuria, ulcerative stomatitis, and epistaxis.

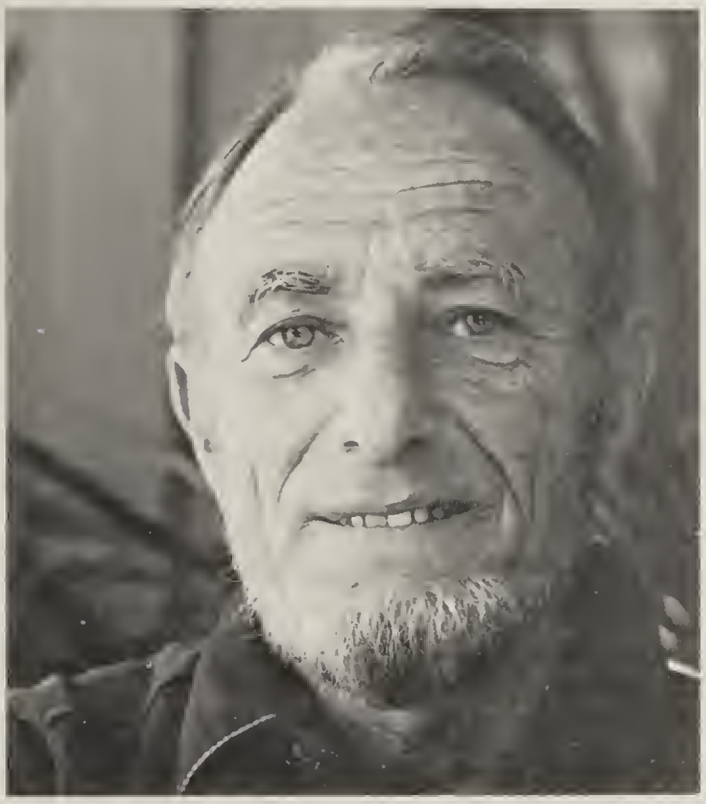
Supplied: Capsules containing 25 mg indomethacin each, in single-unit packages of 100 and bottles of 100 and 1000; capsules containing 50 mg indomethacin each, in single-unit packages of 100 and bottles of 100.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

MSD MERCK SHARP & DOHME

President's Page

HEALTH PROFESSIONS EDUCATIONAL ASSISTANCE ACT OF 1976



EDWARD SATTENSPIEL, M.D.

The Health Professions Educational Assistance Act of 1976 (S—3239), introduced by Sen. Kennedy (D-Mass.), has been passed by the Senate and is now before a Joint Conference Committee. This bill includes provisions which, if adopted, would potentially affect every physician in the country. Following are some of the "gems" contained in this latest proposed expansion of Federal bureaucracy, as delineated in the AMA's *Legislative Roundup*.

(1) The Secretary of H.E.W. would be required to compile, collect and analyze data pertaining to all physicians and dentists. He would also collect information on all medical students annually.

(2) A Study would be authorized by the Secretary of the costs of teaching and providing chiropractic services with a view to determine the supply of chiropractors and the amount of capitation grants which might be provided to chiropractic schools.

(3) Federally insured loans to medical students of up to $10,000. per year per student would be established. These could be paid off by Secretary if the student agreed to serve in the National Health Services Corps. or in private practice in a shortage area for one year for each year of obligation assumed by the Secretary.

(4) The Secretary would annually designate shortage areas and would assign personnel. *Present requirement of certification of need by state and local medical society would be dropped.* Any person breaching his contract to serve in a shortage area would be obliged to pay back *three times* the amount of the funds received.

(5) A National Council on Postgraduate Physician Training would be established by the Secretary of H.E.W. This council would recommend the national total of post-graduate training positions by specialty and subspecialty. Not less than 57% of all positions available would be in family practice, general internal medicine, general obstetrics and gynecology. Post-graduate physician training control provisions would *not* apply to osteopathic physicians.

(6) Capitation grants to medical and dental schools would be made annually by the Secretary, provided that in 1978, 1979 and 1980, — 25%, 30% and 35% respectively of all students enrolled agree to accept National Health Service Corps. scholarships. Similar capitation grants would be made to schools of veterinary medicine, optometry, pharmacy, pediatry, and public health but *without* the service requirements or percentage limitations or controls.

(7) The Secretary would, within two years, develop and establish model standards for licensure, *prepare examinations for licensure and relicensure*, and develop standards

for continuing education. Standards would include *satisfactory performance on tests.*

Here, then is a bill which, if passed, provides for one federal appointee, the Secretary of H.E.W., to determine who will practice medicine, how he will practice, where he will practice, and whether he may continue to practice. Can you hear the echoes of George Orwell's *1984* as loudly as I can? How do we, the medical profession, as the most concerned group, combat the implications of this legislation? The only effecive spokesman we have on the national scene is our own national organization, the AMA, and you can be sure that the AMA has been struggling against this octopus since its inception. Voluntarily or mandatorally, we need a strong national organization like never before! To those who remain unconcerned, my sympathies.

Dean's Page

THE ARIZONA PERINATAL PROGRAM

NEAL A. VANSELOW, M.D.

In the summer of 1975, The Robert Wood Johnson Foundation of Princeton, New Jersey announced the award of one-time, five year grants to eight regional programs which were designed to reduce maternal and infant mortality, birth defects, and other disabilities of the mother and newborn. One of the grants was made to the State of Arizona with Dr. Joseph Daily of Phoenix and Dr. C. D. Christian of our Department of Obstetrics and Gynecology as co-directors of the project. The grant was made jointly to the State of Arizona and the University of Arizona and is funded through the Arizona Medical Association Foundation.

Of the one-hundred plus initial applicants, Arizona was selected largely because of the spectacular reductions in perinatal mortality that had been accomplished by the Arizona Infant Transport Program. Building on this experience the Division of Reproductive Medicine at St. Joseph's Hospital of Phoenix and the Department of Obstetrics and Gynecology at UAMC have begun to transport high-risk gravidas to their labor and delivery units from all reaches of the State. Patients are transported by helicopter, fixed-wing aircraft, automobile or ambulance, whichever is the more appropriate and available method.

The physicians of the State now have access to telephone consultation from either hospital and the ability to transport the high-risk gravida to a central hospital (St. Joseph's — Phoenix or UAMC — Tucson). Both hospitals have a full range of medical and hospital services and highly specialized staffs, and facilities to manage the most serious and complex risks and problems of the pregnant woman and the newborn.

The grant (which is allocated to partially offset transportation and hospital costs, but in no way can cover all attendant costs) is unique in the sense that it was made jointly to the State and the University. We are proud to be a part of such a joint effort — not only for the intrinsic good that it provides, but because it allows the College of Medicine and St. Joseph's Hospital the Opportunity to work hand-in-hand with practicing physicians in Arizona to reduce maternal and infant morbidity and mortality.

The program is "regional" in the sense that it is for a region (in this case the entire state) but it is not regional in the sense that it mandates transport of patients from a certain area to a specified center. Any physician from any part of the state may transport a mother to either Phoenix or Tucson. Referral patterns are developed along those lines that are most appropriate to the needs of the patient and the physician rather than by geographical boundaries.

At the present time the Arizona Medical Center is receiving approximately 20 maternal transports a month with an additional six to eight going to Phoenix. Since the transport program began there have been some 400 such patients at UAMC. The Department of Obstetrics and Gynecology is currently reviewing the computerized data on these patients as to maternal and fetal outcome.

The impact of this program has already been great and we are very pleased to have the opportunity to work with Arizona physicians in such a direct and meaningful way.

Neal A. Vanselow

AM *Clinical Oncology In Arizona*

COLORECTAL CANCER: DIAGNOSIS, NATURAL HISTORY, AND TREATMENT

NEEL HAMMOND, M.D.
RICK BROWN, M.D.

Editors: Sydney E. Salmon, M.D., Professor and Head, Section of Hematology and Oncology, University of Arizona College of Medicine, Tucson, Arizona 85724; Robert H. Thoeny, M.D., Dir., Radiation Oncology, Good Samaritan Hospital, Phoenix, Arizona 85006; Darwin W. Neubauer, M.D., General Surgeon, 720 Country Club Road, Tucson, 85716.

INTRODUCTION

Colorectal cancer accounted for 99,000 new cases of cancer, the highest of any visceral malignancy, and 49,000 deaths, second only to lung cancer, during 1975. Of those numbers, 600 new cases were Arizonans, accounting for the death of 300 Arizona residents. Even more discouraging is the lack of any great change in the 5-year survival rates by stage of disease, from 32% in the 1940's to 45% in the late 1960's. This paper will briefly review and discuss some of the means of early diagnosis and therapy including new advances in the potential for adjuvant radiotherapy and chemotherapy of colorectal cancer. Since more than 95% of all colorectal tumors are adenocarcinomas, the discussion will be limited to that group.

ETIOLOGY

The epidemiologic pattern of colorectal tumors gives some insight into proposed etiologies. The United States and the Western European countries have high rates of fat and protein intake as well as having higher incidence of colorectal tumors. It has also been found that in countries with more bulk, fiber, or cellulose in the average diet there is a lower incidence of both colorectal cancers and diverticular disease. Thus, it has been conjectured that the rapidly increasing incidence of colorectal carcinoma and decreasing incidence of stomach carcinoma are a consequence of the highly refined diets of the industrial nations.

It is likely that a carcinogen (or carcinogens) is at least partially responsible for the appearance of colorectal tumors. Whether this is in combination with viral, genetic, dietary, or other factors remains an unanswered question.

DIAGNOSIS

Earlier diagnosis and treatment of colorectal cancer is the best means of improving survival rates. By the time the tumor has spread into regional lymph nodes, 5-year survival rates are less than one three with surgery alone, while those of strictly mucosal lesions approach 100%. Patients who are symptomatic at the time of diagnosis have a 5-year survival rate of 49%, whereas asymptomatic patients have a 88% 5-year survival rate.

Early symptoms vary according to tumor location. Right colonic lesions often present with nonspecific abdominal pains or "gas." Another presentation for lesions of the right

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From: Arizona Medical Center, 1501 N. Campbell Ave., Tucson, AZ 85724, Sect. of Medical Oncology (Dr. Hammond) and Sect. of Radiation Oncology (Dr. Brown) colon is unexplained iron deficiency anemia. With otherwise unexplained peptic-ulcer-like symptoms, a barium enema is indicated. The most common symptom of left-sided colorectal tumor is a minor or major change in bowel habits. Bleeding is another frequent symptom of left-sided tumors, although massive or bright red bleeds are more frequently due to diverticulitis. Unfortunately, late signs of tumor are often the presenting symptoms. Aside from symptoms of distant metastases, late regional symptoms include tenesmus and abdominal masses. Tenesmus in rectal adenocarcinoma is a late symptom indicating a minimum of nerve infiltration, if not regional node involvement. One unusual but noteworthy late presentation for descending colonic carcinoma is perinephric abscess secondary to perforation. Symptoms seem to be determined by function-absorption for the right colon and cecal areas, and evacuation for the left colon and rectosigmoid areas.

By screening of the asymptomatic patient, one would hope for early detection of colonic tumors. Routine physical exam should include not only a rectal, but bimanual exam in both males and females. This detects at least 40% and probably a much larger percentage of all colonic tumors. Sigmoidoscopic exam, though uncomfortable and time-consuming (as well as expensive for mass screening), is capable of detecting well over one half of all colonic tumors.Unequivocally, this exam is valuable in any patient with even minor symptoms. Cytologic exam of the stool has been shown to be fairly accurate, but is cumbersome. Mass screening by tests for occult blood has been proposed. If this type of screening is to be used, one should refer to Greegor for technique to avoid pitfalls.

Another method for detection of colonic tumors is the barium enema exam. Sigmoidoscopic exam should always precede a barium enema, except under unusual circumstances. Inconvenience, cost, and discomfort make the barium enema an impractical method of screening asymptomatic patients.

Colonoscopy currently has no role in screening because of inconvenience, cost, and a low (but significant) complication rate. The current uses of colonoscopy are clarification of the nature of lesions noted on barium enemas and removal of benign adenomatous polyps, especially in patients who are poor surgical risks.

The identification of groups at risk would assist in any screening effort. Advanced age is known to be a risk factor, with a mean age at diagnosis greater than 65 years and peak frequency in the eighth decade of life. However, we do not advise routine barium enemas or colonoscopies simply on the basis of age.

First-order relatives with a risk factor threefold greater than the general population, ulcerative colitis, familial polyposis, villous adenomas, and adenomatous polyps are existing conditions that should be considered indications for colonoscopy and barium enemas.

STAGING

The extent of cancer is of major importance in the planning of treatment, especially therapy. Most metastases are to the regional lymph nodes, liver, and lungs, with bone, brain, skin, and peritoneal cavity being less frequent, but clinically apparent, areas of involvement.

The incidence of local recurrence is greatest in rectal tumors, being 23%-34% below the peritoneal flexure and less than 10% above. Gunderson has demonstrated the close correlation of extension through the serosa, with or without positive lymph nodes, with marked increase in local failure. Death comes to patients as a consequence of either local complications (50%-75%) and/or distant disease (25%-50%).

The purpose of a staging system is to determine extent of disease and predict survival rates as a function of a given treatment. Most efforts at staging have focused on the following prognostic signs: degree of anaplasia, wall penetration, location of the lesion, lymph node involvement plus the number of lymph nodes actually involved, venous invasion, perineural involvement and pre-operative obstruction.

The Gunderson-Sosin modification of the Astler-Coller system (shown in Table 1) takes into account more of the major prognostic factors than previous systems. Described in conjunction with a careful analysis of data obtained previously by Wangensteen in a series of second-look operations, it does not have the drawbacks inherent in systems based on clinical staging or end-result autopsy data. It contains the most homogeneous groups of any currently active staging system and appears to have the most predictive value. (Although not shown in Table 1, the C^1 category is also divided into subcategories on the basis of *number* of nodes

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Table 1

STAGING SYSTEMS FOR COLORECTAL CARCINOMA

Comparison of scheme with a modification by
Gunderson and Sosin of the Astler-Coller system

System		
Duke's	**modified Astler-Coller**	**Description**
A	A	Nodes negative; lesion limited to mucosa
	B_1	Nodes negative; extension of lesion through mucosa but still within bowel wall
B	*B_2	Nodes negative; extension through the entire bowel wall
C	C_1	Nodes positive; lesion limited to bowel wall
	*C_2	Nodes positive; extension of lesion through the entire bowel wall

*Separate notation is made regarding degree of extension through the bowel wall; (microscopic only [m]; gross extension confirmed by microscopy [m&g] and adherence to or invasion of surrounding organs or structures (B_3, C_3).

†See text for further discussion of this group.

involved (i.e., more than three, less than three). This is based on the finding that regional-local failure is greater than 80% with all B_3, C_2 and C_3 lesions and greater than 50% with B^2(g) lesions and C^1 lesions *with more than three involved nodes.)*

THERAPY

Surgery

The only form of curative therapy currently available is surgery. Once the diagnosis is established, the presurgical work-up should include (1) CEA, (2) colonoscopy, and (3) lymphangiogram, as well as routine liver function studies, chest x-ray, and barium enema. Refinement in surgical procedures and reductions in surgical mortality rates have, to date, made the only gain yet discernible in the treatment of colorectal cancer. The Turnbull "no touch" technique, involving early clamping of vessels and lymphatics, and/or wide resection of lesions have improved 5-year survival rates, most notably in patients with positive lymph nodes. When a patient is going to surgery several things should be remembered. First, the patient should be made aware of at least some of the postoperative possibilities, i.g., colostomy. Of special importance in lesions below the peritoneal flexure are the problems of abdominoperineal resection. Foremost among these problems are impotence in approximately one third of the patients and the extended recuperative period required. When possible, the surgeon should try to determine the type of procedure needed prior to surgery. This is sometimes difficult, especially with lesions below the peritoneal reflection yet more than 5-7 cm above the dentate line. This location may allow a low anterior resection or require an A-P resection. Sphincter-sparing proceedures have not been technically satisfactory to date. For a low anterior resection to be considered adequate, a 2-cm tumor-free margin should be obtained and wide pelvic dissection with adequate, node basin accomplished. Preoperative antibiotic bowel prep is currently felt to be of use. Omentectomy and oophorectomy are probably warranted if feasible when free perforation is noted as surgery. Second-look procedures because of failures to detect tumor and remove tumor curatively have been abandoned as curative measures. In an advanced age group, the surgical mortality rate is about 4%.

Adjuvant Radiation Therapy

Combining surgery and irradiation is attractive for patients with rectal carcinoma who are at high risk for local failure (i.e., B_2[g], B_3, and all C's). We would prefer to remove all gross disease with an operative procedure and utilize radiation for microscopic or subclinical disease. The reason for this approach is two-fold: (1) post-operative irradiation would not interfere with surgical decisions regarding anterior resection vs. abdominal-perineal, partial bladder resection, extent of node dissection, etc., (2) with radiation alone, the bulk of tumor would require higher doses preventing a suitable therapeutic ratio between cure and complications.

Utilzing the above logic and Gunderson's review of Wangensteen's data (as described previously in this paper), one of us (RB) participated in a pilot study of post-operative irradiation in rectosigmoid carcinoma instituted at the Latter Day Saints Hospital in Salt Lake City in 1971. As of December 1975, 46 patients had been entered into the study; 31 were referred after "curative" operation but were at high risk for local regional failure (Table 2).

Table 2
POSTOPERATIVE RADIATION THERAPY FOR COLORECTAL CARCINOMA: PATIENT GROUP

(44 patients had operation and external radiation; 2 patients had external radiation and implant after recurrence*)

Clinical Presentation	No. pts.
1. After first operation+	36
a) Curative (no known residual)	31(30)**
b) Resection but residual	5 (6)**
2. After recurrence+	10
a) Gross recurrence removed	4
b) Resection but residual	4
c) Without removal*	2

**-In patient M.S. the question of residual tumor was raised on small bowel and barium enema films taken during the course of radiation but this was not documented.

+2 patients in the primary group and 3 in the recurrent group had distant metastasis at the time radiation was given for an attempt at permanent control of the local-regional area.

The techniques of irradiation were individualized based on the type of operative procedure, area and extent of initial tumor involvement, and position and degree of mobility of small intetine. Figure 1 shows the radiation portals encompassing the sites of failure found in the Wangensteen second-look series. The extended fields were treated at a rate of 850-900 rads per week to a total of 4500 rads/five weeks to 5100 rads/six weeks.

Absolute survival rates at six-month intervals are shown in Table 3. All of the cancer-related deaths have occurred at less than two years. This preliminary data suggests post-operative radiation has an excellent chance of preventing local failure in patients who are at high risk. Distant metastases occurred subsequently in 8/34 (23.5%) in the primary group and 3/8 (37.5%) in the recurrent group. Apparently, as combined sur-

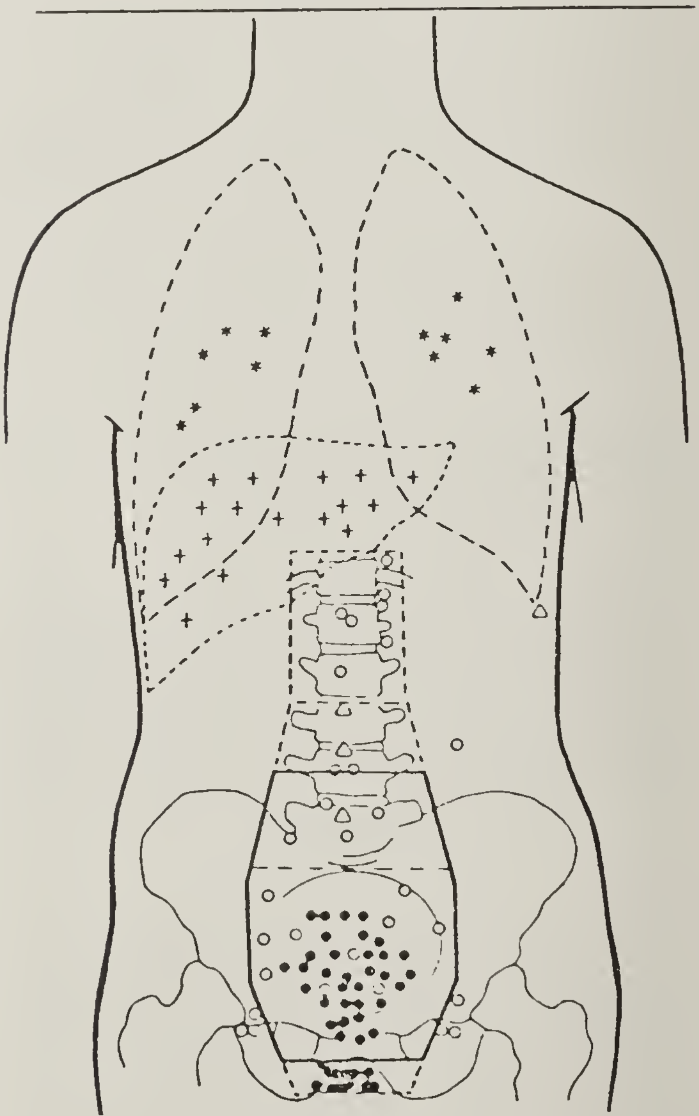

Figure 1

Patterns of failure in the 52 patients with evidence of carcinoma after initial operative procedure—superimposed radiation fields (mid L^4, L^3, T^{12}) Legend: (Each marking indicates a single instance of such failure occurring alone or as any component except for lymph node failures where each major area of involvement is indicated.) * = lung metastasis; + = liver metastasis; △ = wound implant; ● = local failure in pelvic or perineal tissues; ○ = lymph node involvement—Only the superior extent of aortic nodal failure is indicated. One patient had a nodal mass in the colostomy mesentery. (From Gunderson and Sosin [reference 3]. Reproduced with permission from the authors.)

Table 3

POSTOPERATIVE RADIATION THERAPY FOR COLORECTAL CARCINOMA: SURVIVAL RATES

(From time of operative intervention — 44 patients; From onset of radiation — 2 patients without an operation)

Clinical Presentation	No. of pts.	Survival Rates (absolute) 6 mo	12 mo	18 mo	24 mo	30 mo	36+ mo
After 1st operation*	36	30/31 96.8%	19/20 95%	13/16 81.3%	6/11 54.5%	3/6 50%	2/4 50%
Rectum	17	14/15	8/9	6/7	1/3	—	—
Rectosigmoid	8	7/7	3/3	1/2	1/2	1/1	1/1
Sigmoid	5	4/4	4/4	3/3	2/3	1/2	1/1
Other Colon	6	5/5	4/4	3/4	1/3	1/3	0/2
Recurrence*	10	8/8	5/6	2/4	0/3	—	—

* 2/36 and 3/10 had disease outside of the radiation field at the onset of treatment (4-hematogenous; 1-omental).

gery and irradiation increase local control, early eradication of distant metastases with systemic therapy assumes increasing importance.

Systemic Therapy for Advanced Disease and as an Adjuvant to Local Therapy

Chemotherapy of advanced metastatic colorectal tumors remains unsatisfactory. 5-Fluorouracil has been the most widely used drug. Most oncologists agree that 17%-20% of patients with widespread metastatic disease show objective evidence of tumor regression. Responders to 5-FU alone have a median survival of 17 months compared to approximately 7 months among non-responders and those patients with metastatic disease who do not receive treatment. Response appears to be best in younger patients, in patients with longer disease-free states, and in patients with metastatic sites of involvement other than the lung. Evaluations of 5-FU have included studies of the route of administration, schedule of administration, and combination of 5-FU with other agents. The currently accepted mode of administration is the intravenous route. Oral therapy gives decidedly shorter durations of response in both hepatic and other areas of metastasis (22 vs. 8 weeks), and the frequency of response is probably also lower. Intra-arterial 5-FU infusions are not generally used, although one recent report describes a 55% response rate in previous treatment failures given high-dose intrahepatic arterial infusions. The finding that methyl-CCNU is equivalent to 5-FU as a single agent prompted trials with 5-FU plus nitrosoureas. Successful combinations to date include the following: (1) Moertel at the Mayo Clinic using vincristine, 5-FU (i.v. Days 1-5 q 36 days), and methyl-CCNU found a 43.5% response rate compared to 19.5% with 5-FU alone. (2) The Southwest Oncology Group, comparing weekly i.v. 5-FU, with or without methyl-CCNU, found the nitrosourea combination to be superior with 30% vs. 14% response rates. (3) Falkson, using a combination of vincristine, imidazole carboxamide, BCNU, and 5-FU, compared with 5-FU given monthly for five-day courses, again found the combination to be superior—43% vs. 25%. Most importantly, the survival of treated patients will hopefully be increased. Although there are negative reports with 5-FU-nitrosourea combinations, there appears to be an advantage to the combination of 5-FU with a nitrosourea. These combinations have some promise for use in adjuvant chemotherapy after surgery or surgery plus radiotherapy. However, they must currently still be viewed as investigational approaches.

Adjuvant chemotherapy with 5-FU alone, either intraluminally or parenterally, has been uniformly ineffective with the doses and schedule used. Now that staging has been standardized to a greater degree, and with the advent of combination chemotherapy for colon cancer, studies will be more carefully designed and results may be better. The Gastrointestinal Study Group is currently investigating the combination of methyl-CCNU and 5-FU, and there are at least two ongoing adjuvant studies including chemo-immunotherapy. An M. D. Anderson study compares the effects of BCG given by scarification vs. BCG plus 5-FU. A Southwest Oncology Group Study, compares methyl-CCNU plus 5-FU vs. methyl-CCNU plus 5-FU plus oral BCG. Both of these studies are for Dukes' C lesions, although the Southwest Group is also including patients with involvement of the entire thickness of the muscularis but no positives nodes. Results have been reported only in the first of these studies and are not conclusive.

We believe that the future approach to colorectal cancer treatment will include both effective local therapy with surgery and radiotherapy, as well as adjuvant chemotherapy or chemoimmunotherapy for micrometastases. Proof of the value of this approach will require years of study, but this is a worthwhile endeavor for major oncology centers.

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Seminars in Chest Medicine

OAT CELL CARCINOMA OF THE LUNG

D. Hood Franks, M.D.

This continuing series of articles entitled "Seminars in Chest medicine" will attempt to keep the reader abreast of developments in the broad field of pulmonary diseases. The format used will be that of brief succinct reviews written by the editors as well as guest contributors. Areas of controversy as well as practical chest medicine will be explored. We hope that these reviews will be of value in promoting continuing education for certification examinations as well as a forum for new and controversial issues. The editors welcome comments and discussion from our readers.

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In 1976, 90,000 persons will develop cancer and of them, 81,000 will ultimately die of their disease. This represents a 20% increase in incidence and mortality since 1972, and follows a trend that makes lung cancer a problem of near epidemic proportions.

The epidemiologist may group together the four histologic varieties of lung cancer, but the clinician should consider them as distinct disease entities. Their biologic characteristics and behavior patterns vary widely and often predictably.

Unique among the lung cancers are those 20% called small cell undifferentiated or "oat cell" carcinoma. They are thought to derive from a different precursor cell, perhaps of neuroectoder-

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mal rather than foregut origin.[1] Small cell carcinoma is associated with poor host responsiveness, early widespread dissemination and relatively short survival times. "Paraneoplastic syndromes" with organ dysfunction not directly related to metastatic spread are frequently seen. The initial response to radiotherapy and chemotherapy is greater than that seen in other bronchogenic carcinomas.

The patient almost invariably smokes cigarettes, usually heavily. Only the oat cell carcinoma shows a dose response relationship to cigarette smoking.[2] Symptoms are usually of short duration because of the rapid growth rate of the tumors; more than half the patients are diagnosed within two months of the onset of their first symptom.[3] A strong male predominance persists, but during the past fifteen years the prevalance of women with lung cancer has greatly increased.[4]

Common presenting symptoms include cough, hemoptysis, vague chest pain, shortness of breath and respiratory infection because the frequently centrally located tumor produces endobronchial irritation and encroachment upon the bronchial lumen. These symptoms can be difficult to evaluate in heavy smoking patients who have chronic respiratory complaints. Subtle changes in the pattern and character of the symptoms should be sought and throughly evaluated, especially if associated with weight loss.

Mediastinal lymph node involvement is characteristic of oat cell carcinoma and symptoms often relate to compression of mediastinal structures. Presistent hoarseness suggests left vocal cord paralysis due to compression of the left recurrent laryngeal nerve. Indirect laryngoscopy may provide a diagnostic clue. Esophageal compression leads to dysphagia. Dyspnea may partially relate to diaphragmatic paralysis resulting from phrenic nerve injury. Physical examination may suggest an elevated, immobilized diaphragm which can be confirmed radiologically.

The most impressive manifestation of mediastinal tumor is the superior vena caval syndrome. It results from compromise of the vessel lumen, thereby retarding venous drainage from the head, neck, upper extremities, and upper thorax. Symptoms include headache, vertigo, nasal stuffiness and swelling of the face, arms and hands. Physical examination confirms the presence of edema and collateral venous circulation across the chest. This syndrome is frequently the result of an underlying neoplasm, with oat cell carcinoma being the most common. Radiotherapy and chemotherapy are effective forms of treatment.[5]

Because of a propensity for early metastasis, oat cell carcinoma may present as a metastatic lesion. Bone pain with or without a pathologic fracture and central nervous system involvement are frequent presentations.

The pelvis, ribs and vertebrae are common sites of symptomatic bony metastases. Low back pain is a relatively common complaint of the general population and seldom commands appropriate attention unless a pathologic fracture occurs. Thoracic spine pain is uncommon. The ribs buttress and support the thoracic vertebrae, and pain in this area usually signifies a serious underlying disease. Thus the presence of high back pain in a smoker should stimulate a careful search for its cause with the possibility of an occult lung cancer being at least one consideration.

Oat cell carcinoma may present as one or more expanding intracranial masses. Symptoms include increasingly frequent and severe headaches, nausea and vomiting, personality changes, and seizures. Physical agents placed in the pleural cavity are often ineffective in controlling the effusion. Treatment, therefore, should be directed at the centrally located lesion utilizing radiotherapy and/or chemotherapeutic agents.

Diagnostic evaluation begins with a chest x-ray and culminates in histologic proof of the underlying neoplasm. The chest x-ray usually shows a hilar prominence or perihilar mass (3/4 of all patients).[7] Only 1 in 10 patients will have a peripheral mass as the sole chest x-ray abnormality. Mediastinal fullness is a relatively common finding and tomography may be helpful.

Sputum cytology will be positive in two-thirds of patients with oat cell carcinoma;[8] the diagnostic yield increases from 45% positive on the first specimen to 86% positive by the third specimen and to 96% positive when five specimens are examined.[9]

Fiberoptic bronchoscopy is usually diagnostic except when the tumor extends along the submucosal plane blocking access to the distal primary lesion. In this situation mediastinoscopy is a very useful alternative. Positive biopsies will be obtained in 70% of patients with oat cell carcinoma so evaluated.[10] This procedure has largely replaced the scalene node biopsy.

Once the diagnosis is established, the extent of tumor spread must be determined. If the tumor is regional, (i.e. confined to a hemithorax and adjacent lymphatics which can be encompassed in a single radiotherapy field), radiotherapy is begun with a curative goal. Systemic disease is often treated with chemotherapy alone, reserving radiotherapy for the palliation of symptomatic metastatic lesions. Findings may consist of motor deficits, or lethargy and coma with focal neurologic signs. Metastatic lesions are seldom single and the neurological examination may be confusing. The recognition of this syndrome may prevent unnecessary craniotomy since these patients can be adequately managed with corticosteroids and radiation therapy.

Careful physical examination may reveal lesions which are accessible for biopsy and identify problems requiring specialized treatment. Subcutaneous nodules and supraclavicular lymphadenopathy are frequently overlooked sources for histologic diagnosis. Examination also provides staging information. Special attention is given to the common sites of metastasis such as the brain, bone and liver. Palpation of skeletal structures for tenderness may suggest areas to be radiographically examined.

Certain physical findings are conspicuous in their absence in patients with oat cell carcinoma. Digital clubbing is uncommon and hypetrophic pulmonary osteoarthropathy is rare. The latter is a syndrome of symmetrical proliferating subperiosteal osteitis with new bone formation and periosteal thickening. It normally affects the wrists and ankles and occurs in 4-12% of patients with bronchogenic carcinoma. In one series of 60 patients with lung cancer and hypertrophic osteoarthritis, no small cell carcinoma was encountered.[6]

Because of their central location, oat cell carcinoma infrequently involves the pleural surface. The rare effusion resulting from lymphatic and/or venous obstruction is usually cytologically negative.

Evaluation for metastases focuses on frequently involved sites such as the skeletal system, liver, and brain. Unsuspected bony metastases are frequently identified with bone marrow biopsy of the posterior iliac crest. Even in the absence of bone pain, hematologic abnormalities, or elevated alkaline phosphatase, bone marrow extension can be documented in 40-50% of all patients evaluated in this manner.[11, 12] Bone scans in this instance are much more sensitive than x-rays, but are positive in only 70% of patients with osseous metastases. However, 19% of patients with negative bone marrow biopsies have positive bone scans.[13] Thus, the two procedures are complementary.

Liver and brain scans round out the evaluation of possible systemic spread. False positive liver scans can occur in patients with Laennec's cirrhosis, hepatocellular necrosis,hepatic steatois and granulomatous hepatitis.[14] Therefore, a patient with a "positive" liver scan and no other evidence of systemic spread may require a liver biopsy to select appropriate therapy.

Brain scanning has recently been augmented by computed axial tomography,[15] a non-invasive procedure with diagnostic accuracy approaching that of cerebral angiography and pneumoencephalography. In selected situations, when the index of suspicion of metastatic extension is high, both brain scan and computed tomography should be utilized.

In rare instances, thoracotomy may be required for diagnosis. However, most surgeons no longer consider this tumor to be operable since only 6% of patients judged clinically resectable will survive two years.[16] Dissemination, often not yet detectable, has occured in most patients at the time of presentation. Those few patients who have truly localized disease are adequately treated with radiation therapy alone. Studies comparing the two methods yield patients surviving five years only in the group treated radio-therapeutically. The median survival rate is also longer in the radio-therapy group.[17]

Small cell carcinoma is the most responsive of the lung cancers to a variety of chemotherapeutic agents. This may be related to the large number of cells actively dividing (growth fraction) among the total number of cancer cells present. Unfortunately, the development of resistance is also rapid. Single agent chemotherapy has been superseded by combination chemotherapy to capitalize on different modes of cytotoxic action. Ideally, overlapping toxicities are avoided. The result is an increase in tumor cell killing and a reduced rate of resistance. Utilizing such programs, median survival times have increased to more than thirty weeks, even in patients with extensive disease.[18] This represents a marked increase over the seven week survival of patients treated with a placebo and the seventeen week survival following treatment with a single effective agent such as cytoxan.

Recent efforts to control regional disease have combined chemotherapy programs with radiotherapy. Radio-therapy is superior for control of the primary lesion, while chemotherapy controls the microscopic metastases outside the field of irradiation. Chemotherapy is delivered for one or two courses followed by radio-therapy and then repeat chemotherapy. Median survival times approaching one year are achieved by utilizing such programs.

When the diagnosis is established and a treatment program is chosen, the physician is confronted with additional challenges. Each new symptom may indicate a metastasis and should be investigated carefully. No headache or backache can be considered trivial, but alone it does not necessarily indicate treatment failure and require a change in chemotherapy or the institution of palliative radiotherapy. As in other malignancies, steady progression of a given symptom is a significant finding. It often results from an expending metastasis with progressive destruction of normal tissue. Pretreatment x-rays and radionucleide scans are particularly valuable because serial tests may confirm the clinical impression of advancing disease.

Oat cell carcinoma is frequently associated with "paraneoplastic syndromes". These tumors are capable of producing peptide hormones which may cause severe metabolic disturbances; limb weakness and mental confusion can be misinterpreted as evidence of advanced metastatic disease. Hypokalemic alkalosis and profound proximal muscle weakness may be traced to the ectopic production of ACTH. The high corticosteroid output by the adrenal gland produces renal potassium wasting, hypertension and hyperglycemia. Other findings of Cushing's syndrome such as the centripetal obesity, moon facies and striae are usually absent because of the rapid progression of the disease. Treatment aimed at the primary tumor is the most effective therapy. If this fails, metyrapone and aminoglutetamide can be administered to block adrenal hormone synthesis and alleviate the symptoms.[19]

Another common problem results when antidiuretic hormone (ADH) is produced by an oat cell neoplasm. Severe hyponatremia in the absence of renal or adrenal disease results in progressive weakness, confusion, and sometimes seizures and coma. Water intoxication is best treated by water restriction. The infusion of saline or other salt containing solutions may cause clinical deterioration. As in the ectopic ACTH syndrome, optimum management includes treatment of the underlying tumor to decrease its cell mass and thereby reduce the amount of ADH secreted.

Hypercalcemia rarely occurs in patients with oat cell carcinoma,[20] in spite of extensive osseous metastases. Hypercalcemia is usually associated with squamous cell carcinoma of the lung. It affects 8-22% of patients with squamous cell carcinoma and results from production of a parathormone-like substance by the tumor. Hypercalcemia is one of the few hormonal paraneoplastic syndromes in lung cancer not associated with oat cell carcinoma.

Non-metastatic neurologic manifestations are relatively frequent in patients who have carcinoma and occur six times more often in this cell type (56%) than in a randomly chosen group of patients with lung cancer (9%) (21). The syndromes include dementia associated with diffuse slowing of the electroencephalogram, peripheral neuropathy (sensory and motor), and subacute or acute cerebellar degeneration with bilateral symmetrical ataxia. The Eaton-Lambert syndrome with its myasthenia gravis-like myopathy is seen almost exclusively in patients who have an oat cell tumor. These syndromes can be the first evidence of an undiscovered neoplasm. Extensive disease need not be present and months may elapse before a lesion can be identified on chest x-ray.

Research on the treatment of small cell carcinoma emphasized the importance of evaluating and comparing combinations and schedules of agents such as cytoxan, methotrexate, CCNU, adriamycin, and procarbazine, all of which have been proven effective as single agents. Since patients will initially respond to combined modality programs, treatment failures due to central nervous system metastasis are becoming more commonplace. This presumably results from the inability of the drugs to cross the blood-brain barrier. Studies of prophylactic central nervous system irradiation are under way to solve this problem.

Finally, little research has been performed on the immunotherapy of small cell carcinoma. Because of the low level of antigenicity found in these tumor cells, they seem to be poor candidates for immunologic control. One study nevertheless did show an improved response with the addition of *Corynebacterium parvum* immunotherapy to a standard chemotherapeutic regimen.[22] However, the author has suggested that the immunotherapy increased marrow tolerance allowing increased doses of the chemotherapeutic agents to be given to the immunotherapy group. It remains to be seen whether or not this embryonic modality has a place in the ever widening combinations of approaches against this formidable tumor.

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Seminars in Endocrinology and Metabolism

Continuing with the present issue of Arizona Medicine is the series of articles entitled "Seminars in Endocrinology and Metabolism." The purpose of these short review articles is twofold. First, due to the rapid proliferation of new knowledge in the field of endocrinology and the multiple tests available for their evaluation, short, clinically oriented reviews would enable the physician to keep abreast of these newer developments as they relate to their practice. In addition, with great stress being placed on voluntary recertification in many subspecialties, reviews such as they could serve as an authoritative, succinct teaching forum. The editors will endeavor to accomplish these goals by utilizing the talents of practicing physicians as guest contributors to this series. Feedback, both positive and negative, is encouraged in order to help us fulfill these objectives.

MARSHALL B. BLOCK, M.D. Editor

"THE EYES HAVE IT": ENDOCRINE CAUSES OF EYE PATHOLOGY

Earl Nepple, M.D.

It is interesting to observe that there are many endocrine conditions which can produce eye abnormalities. Because some of these changes are diverse, nonspecific and not well-known, it was thought worthwhile to review some of the endocrine conditions known to be associated with eye pathology. Perhaps early recognition of some of these eye abnormalities will help to direct attention to their possible underlying endocrine etiology.

Probably the most frequent of the changes one sees in conditions associated with increasing levels of thyroxine or triiodothyronine are lid lag (which is easily demonstrated by having the

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patient look down and noting that the lids lag behind the globe in this maneuver), and lid retraction (noted by having the patient look upward and noting that the globe lags behind the upper lid on upward gaze). Although lid lag may be found in approximately 10% of normal people, when it is associated with the other eye findings of hyperthyroidism it is easily differentitated. Other physical findings which can be associated with the hyperthyroid state include paralysis of ocular muscles, particularly that of limitation of upward movement and decreased convergence ability. There can be a stare phenomenon as well as swelling of the lids, retrobulbar neuritis, decreased visual acuity due to papilledema, retinal edema, retinal hemorrages or optic nerve damage. When there is actual proptosis of the globe, either unilaterally or bilaterally, exophthalmos is thus described. Measurements with an exophthalmometer are needed to document the presence of protrusion and a value of greater than 18 to 20 mm. beyond the lateral margin of the orbit are considered abnormal. Of all the conditions known to be associated with hyperthyroidism only Graves' disease produces exophthalmos. Other causes of hyperthyroidism, such as the ingestion of excessive quantities of thyroid hormone, do not produce true exophthalmos. Because of the exophthalmos and lid retraction, exposure heratitis due to a wide palpebral aperture can occur. Most of the physical abnormalities noted in hyperthyroidism are relieved by returning the patient to a euthyroid state. However, some of the findings one sees in Graves' disease such as the exophthal- euthyroid state. However, some of the findings one sees in Graves' disease such as the exophthalmos may in actual fact not regress with recovery. Likewise muscle paralysis may require additional therapy if significant fibrosis following edema formation occurs. In fact,exophthalmos has been described in patients who are euthyroid and exhibit no evidence for thyroid disease at the time of initial presentation. Only later do abnormalities in thyroid gland function appear in many of these patients. There are other conditions which have been associated with proptosis and these need to be differentiated from that due to thyroid disease.

Besides tumors of the muscle, connective tissue or nerve located retroorbitally which can produce unilateral exophthalmos, patients with Hashimoto's thyroiditis have been reported to present with exophthalmos. Furthermore, one can get proptosis with Cushing's syndrome, acromegaly, as well as mild cirrhosis of the liver. It is extremely important to differentiate unilateral exophthalmos due to other causes from that due to Graves' disease. When the patient does not have obvious elevated peripheral thyroid hormone levels and does not appear to be thyrotoxic, the differential diagnosis becomes much more difficult. One of the cardinal features of Graves' disease is that the gland functions autonomously, i.e., it is no longer under degative feedback regulation. To demonstrate this phenomenon, a Cytomel (triiodothyronine suppression study can be performed. If the radioactive iodine uptake is not suppressed following triiodothyronine, the gland can be described as autonomous. A newer, not yet clinically available technique to demonstrate this same phenomenon has accrued from studies utilizing thyrotrophin releasing hormone (TRH). This three amino acid polypeptide made in the hypothalamus regulates thyrotrophin (TSH) release from the pituitary gland. It has been demonstrated that patients with unilateral exophthalmos who are euthyroid by all standard parameters have an inability to release serum TSH from the pituitary gland after TRH is administered. It is thought that although peripheral blood thyroid hormone levels are normal, within the hypothalamus-pituitary region, levels are elevated thus turning off the normal responsiveness of the pituitary to TRH.

Another endocrine condition that can cause significant eye pathology is diabetes mellitus. Obviously the severe retinopathies which one sees in diabetes mellitus can lead to gross intraocular hemorrhage and blindness. Although extroocular muscle paralysis can also occur unilaterally of bilaterally in diabetes mellitus, an intracerebral cause, i.e., tumor, always needs to be excluded before ascribing the abnormality to diabetes mellitus. The wide spectrum of changes one can see in diabetic eyes has led many to ask what is the cause for the retinopathy. At the present time the answer is not at hand but there does seem to be a body of evidence developing that control of blood sugar may be of significant value in limiting the progression of severe diabetic retinopathy (see June, 1976 ARIZONA MEDICINE).

An occasional patient with calcium metabolism abnormalities can be seen with significant eye symptoms and findings. Patients with hypercalcemia can present with the deposition of calcium crystals in the cornea and/or conjunctivae. For instance, diffuse deposits in the subepithelial layers of the cornea with diffusely hyperemic conjunctivae are see in the milk alkali syndrome. In band keratopathy, deposits of calcium are usually most pronounced in the palpebral fissure near the limbus and may appear as a horizontal grayish band with dark "holes". Band keratopathy may be seen in severe anterior uveitis, sarcoidosis, multiple myeloma, renal disease, or in Vitamin D intoxication, as well as in 5% or less of patients with primary hyperparathyroidism. Treatment of this condition obviously depends on the underlying cause but corneal epithelial removal using a chelating agent such as EDTA has been tried. However, if the underlying cause is not corrected, calcium deposition may recur. In contrast to the hypercalcemic states, hypocalcemia which can be seen in idiopathic hypoparathyroidism, may lead to the bilateral subcapsular cataracts. Although not specific for hypoparathyroidism, their presence helps in the diagnosis of the idiopathic and pseudo-idiopathic varieties of this disorder. In addition papilledema has been reported in hypoparathyroidism. The presence of seizures and papilledema usually leads to a search for a space-occupying central nervous system lesion, but a patient with hypoparathyroidism may present in such a fashion. Probably all children who present with a seizure disorder should have serum calciums measured before longterm treatment with anticonvulsant therapy is undertaken.

The most prominent iatrogenic cause of cataracts is probably the chronic administration of various cortisol-derived drugs. Longterm administration of pharmacological doses of cortisone or its derivatives can produce cataracts. Although physiological quantities of cortisol usually do not, patients with longstanding Cushing's syndrome may very well demonstrate if left untreated for long periods of time, such lesions.

Space-occupying lesions of the pituitary gland may present with visual complaints well before the resulting endocrine problems become manifest. Approximately one-third of patients with brain tumors of all types present with eye symptoms. Recently a study found that 63% of patients with pituitary tumors presented with visual symptoms and 18% with endocrine problems. Of the visual complaints, uniocular blurred vision was the most frequent. Poor side vision from hemianopia was a relatively unusual complaint. Obviously chiasmal compression due to a pituitary tumor can be demonstrated in 90% of patents by doing visual field determinations. Seventy precent of intrasellar tumors exhibit bitemporal defects and 30% show various other deficits. Field defects from a chiasmal syndrome, i.e., early pituitary tumor applying pressure to the optic chiasm, usually begin in the superior temporal field and progress inferiorly. Early diagnosis of potentially serious endocrine problems such as Cushing's syndrome, hypopituitarism, acromegaly, can be improved by routinely performing confrontation visual fields and ordering appropriate tests. Adequate evaluation of a patient presenting with decreasing visual acuity and no intraocular cause should include fine fisual fields, color testing, color perception testing, skull films and sequential examinations. In terms of numbers, a chromophobe adenoma presenting with hypopituitarism as its endocrine manifestation is the most common pituitary lesion presenting with ocular findings.

Other endocrine conditions such as tumors of the adrenal medulla or adrenal cortex producing a pheochromocytoma or Conn's syndrome respectively can make themselves manifest by the development of hypertension and significant hypertensive retinopathy. Furthermore, hypersympathetic tone may produce some of the findings one sees in hyperthyroidism, i.e., lid lag, lid retraction.

In summary then, there are many endocrine conditions which can produce eye signs and symptoms in addition to that due to diabetes mellitus. These conditions include pituitary lesions and abnormalities of the thyroid, parathyroid, adrenal cortical and adrenal medullary glands. Perhaps closer attention to these possible subtle changes in the eyes may shed additional light on the patient's overall hormonal status.

Radiology
Case of the Month

RADIOLOGY CASE OF THE MONTH

CASE 13

JOHN C. BJELLAND, M.D.

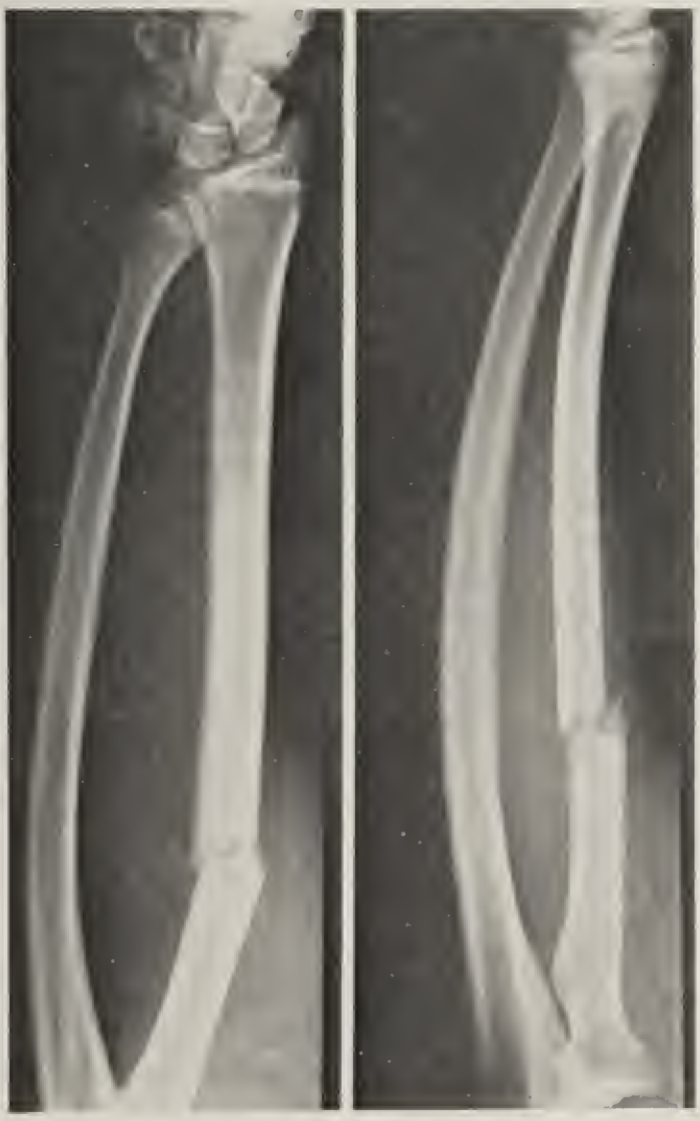

Figure 1

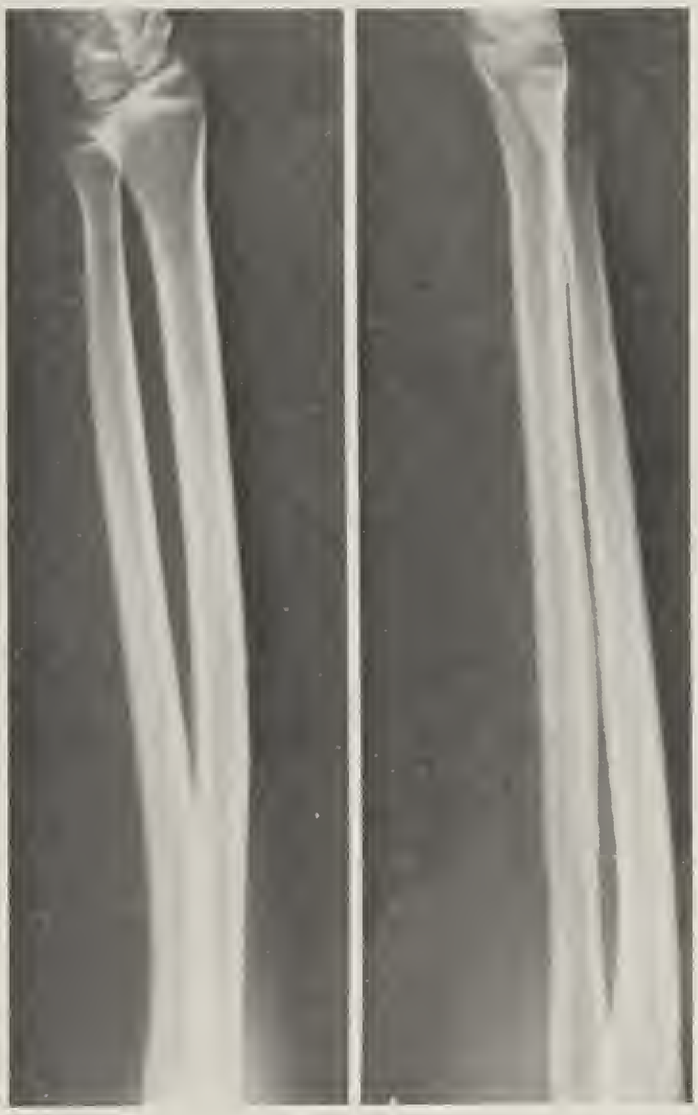

Figure 2

An eighteen year old male sustained a crush injury of his left forearm when it was caught in a dough rolling machine at a bagel bakery. The forearm had forcibly been lodged between the large diameter roller-drum cylinder and feeder belt of the machine. Disassembly of the machine was required to free the patient's extremity. In the emergency room, the forearm was observed to be grossly deformed and swollen, but was free of significant soft tissue or neuro-vascular compromise. The remainder of the patient's medical history and physical examination was noncontributory.

The radiographs in *Figure 1* were obtained to evaluate the obvious clinical fracture of the forearm. However, due to the radiographic appearance of the ulna, comparison views of the opposite forearm were obtained *(see Figure 2)*. The radiographs demonstrate the obvious left radial fracture, but what is the other diagnosis?

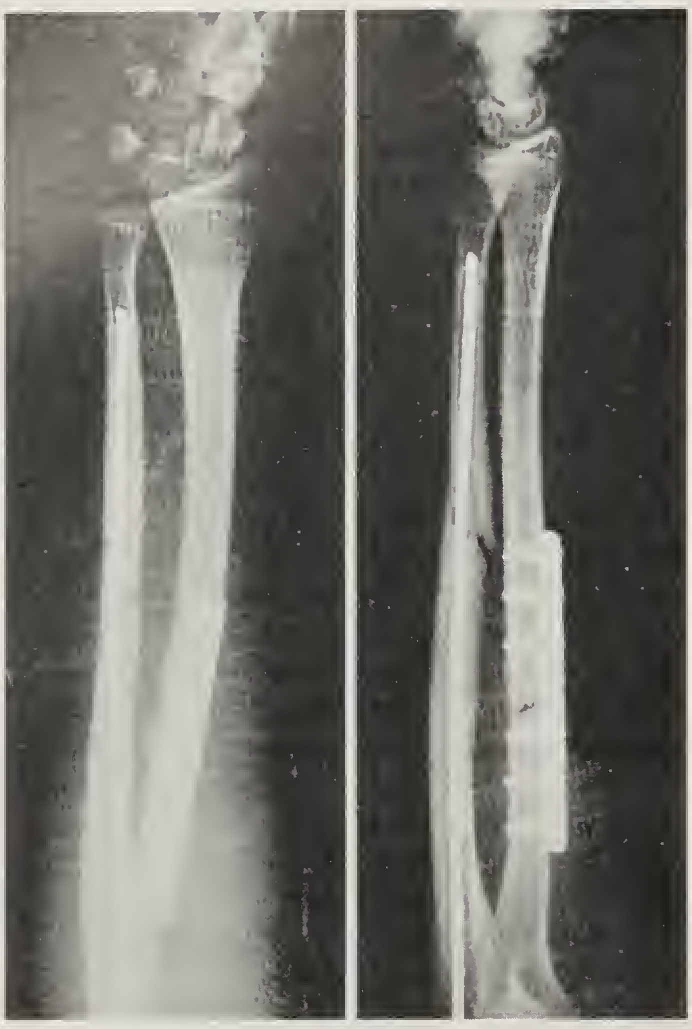

Figure 3: Post operative views of the left forearm.

The radiographs in *Figure* 1 demonstrate a comminuted, angulated midshaft fracture of the left radius and a marked curved or "bowing" deformity of the left ulna. The fractured radius can be attributed to the patient's acute traumatic injury. However, the difficulty in this case lies in correctly evaluating the left ulna. The radiographic differential diagnosis based on its appearance can reasonably be approached from two general etiologic catagories: congenital and traumatic.

The list of congenital entities that can be associated with such a bowed appearance of a tubular bone is a long one. However, most of these entities have bilateral symmetry and the normal contralateral forearm shown in *Figure* 2 contradicts that possibility. Therefore congenital etiologies may be eliminated from the diagnostic gamut. Instead, the traumatic etiologic category should be considered, especially in view of the patient's history of a crush injury.

This case is representative of a recently recognized traumatic entity, termed the "acute plastic bowing fracture of the forearm." This was unrecognized until it was reported by Spencer Bordon at Massachusets General Hospital. In 1970 the prototype of this "bowing fracture" was first incidentally observed in a group of research animals. Then by extrapolation from the results of subsequent *in vivo* and *in vitro* studies, it was postulated that such a fracture could occur in man. Dr. Bordon then collected a series of seventeen such fractures, all of which occurred in a pediatric group of patients. In that group the "bowing fracture" injury was typically sustained when a patient fell on the outstretched arm. The most common radiographic appearance of these fractures consisted of a postero-lateral bowing deformity of the ulna with an associated angulated fracture of the midshaft of the radius (as in this case.)

The acute bowing fracture potential of bone can be attributed to its elastic properties. Due to these properties, when a slowly applied force is exerted on a tubular bone under certain conditions, an elastic deformation can occur in the absence of gross fracture resulting in a "bowed" appearance. Although no gross fracture line is present, microscopically one does observe multiple oblique micro-fractures extending from the medullary cavity of the bone through the full thickness of the cortex in the area of the greatest cortical compression (or area of maximum "bowing").

Of clinical interest is the fact that in Bordon's group, bowed fractures demonstrated a definite tendency to remain bowed, strongly resist closed reduction, and often prevented reduction or relocation of the adjacent associated angulated fractures or dislocations. Due to the presence of this deranged ulnar axis for forearm rotation, the functions of pronation and supination are severly limited. Also, in pediatric patients the unreduced bowed bone has little tendency to undergo the normal remodelling process that such fractures routinely demonstrate. Consequently, the bowed deformity persists even after the numerous micro-fractures are completely healed. Hence, it is of grave clinical import that this fracture never be mistakenly dismissed as a normal variant or congenital anomaly. Instead, it should be accurately recognized as a hallmark of trauma, so proper orthopedic care can be pursuent to the correct radiographic diagnosis.

As regards the orthopedic treatment of this peculiar fracture, the general approach is to complete the "bowed" fracture and then reduce that gross fracture. In the pediatric patient this is achieved utilizing the method of closed, complete fracture with reduction in plaster. However, in the adult such closed procedures have often been unsuccessful. Currently the preferred orthopedic approach to the adult problem is open reduction and internal fixation of the bowed fracture by intra-operatively completing the fracture of the deformed bone and utilizing an intramedullary rod or Rush pin for fixation. Compression plating of the associated angulated fracture (when it is present) is also preferred *(see Figure 3)*.

In conclusion, this case illustrates that, although bone is classically thought of as an extremely rigid, non-yielding structure that sustains gross fractures when excessive force is applied, this is not always true. On the contrary, the physical parameters of force, stress, strain, and, in particular, the temporal interval over which a force is applied to bone all variably interplay in determining the response of bone to either undergo gross fracture or a bowing deformity. The proper radiographic recognition of this traumatic entity has important clinical and orthopedic implications.

ACKNOWLEDGEMENT

Acknowledgement of gratitude for assistance in preparing the patient's history is extended to Ernest L. Gradillas, M.D., Resident, Section of Orthopedic Surgery, University of Arizona, College of Medicine.

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Editorial

PHYSICIAN ADVERTISING

NEIL O. WARD, M.D.

The real reason physicians oppose advertising by and for doctors cannot be easily reduced to glib slogans, but we need to make it obvious to our patients that a future built to the specifications of "consumer advocates" ignores some important lessons of the recent past.

History has taught us that advertisements for "painless", "bloodless" and "guaranteed" cures contributed little, if anything, to medical progress. Testamonials may have sold (and occasionally still do sell) a lot of snake oil but such advertising techniques lack scientific soundness. Acupuncture treatment for deafness is a recent example of a medical ad gone bad.

Efforts to educate the public in good health practices are not to be confused with advertising. Documentation of new and better treatment methods deserves reporting in lay publications (as do potential health hazards), but individual claims of exceptional talents or techniques suggest self-aggrandizement. If our critics find our dignified press code objectionable now, won't they have fun with a diversified advertising mode?!

Advertising weakens our private and personal relationship with patients. The doctor/patient relationship is based on mutual respect and trust. Last Fall, A Lou Harris survey measured public confidence in the leadership quality of 16 different professions and institutions. Medicine scored highest in public esteem with major companies eleventh! Is it logical to assume that our position of public trust will be enhanced by merging medicine with Madison Avenue? Are you concerned about, and, yes, even suspicious of, the "advocates" who would determine the ethical standards of physicians? Those who would program the future with technicians "taking care" of computer card cases have a poor understanding of human nature and no appreciation of what makes American Medicine great. In time of illness and distress we seek help from those we know are concerned with our private needs and personal confidences — not from those concerned with themselves. Pushing a product or a personality has no place in medicine as a profession.

From: "Medical Staff News", Maryvale Samaritan Hospital, Vol III, N. 1, June 1976.

There is no constant correlation between effectiveness of advertising and excellence of the product. If physicians are forced to complete on the basis of extravagant and excessive ads, their more important qualifications will suffer and, in fact, may be lost to patients. If the best-qualified physicians chose the worst advertisers, their practices subsequently may fail economically.

Finally, there is no need for advertising when the very questions a prospective patient poses can be answered authoritatively by the physician's office staff. Many physicians already have printed information sheets available for their patients. The price of a phone call seems a bargain in the best interest of consumerism when compared with the cost, the consumption of paper and the time expended for advertising that may not be up-to-date the day it comes off the presses.

We suspect that the "consumers" are less concerned with this issue than their self-appointed "advocates", but we should reassure our patients that physicians' advertisements are best left in the pages of history.

GOOD (PHYSICIAN'S) MANNERS

M. DAVID BEN-ASHER, M.D.

If the measure of a person is his manners, then we of the medical profession often do not measure up too well. We forget that people do not like physicians as a group, although they do like, even to the point of worship, their own personal physician. Our social behavior as a group often reflects an attitude of arrogance and lack of consideration for others which tends to magnify any underlying distaste for the "rich, fat-cat doctor." There are several areas in which most of us could improve and demonstrate a consideration in respect for others.

Appointments:

With rare exception, there is absolutely no reason for physicians to keep patients waiting one, two and even four hours in their offices prior to being seen. We are not great gurus with whom supplicants are seeking an audience. To the contrary, we are their servants who are here to perform a service for them. The primary causes of long delays in keeping appointments are either unrealistic scheduling or over-booking. If we know that we will never be in the office prior to 10 o'clock in the morning, why start scheduling patients at 9? Over-booking may sometimes be necessary to accomodate people who need emergency care, but it only leads to hurried visits, short tempers, and generally poor care.

The "Beeper"

I have heard radio pagers going off in the middle of concerts, plays and even funerals. This is a modern up-dated version of the not-too subtle form of advertising which used to be carried out by having physicians paged continually by name in public places. In Pima County, this was done away with by assigning call numbers to doctors in the pre-beeper years. Interestingly enough, when we were only called by number, there was a substantial decrease in the number of calls coming through. The harsh whine of the beeper going off is just as and possibly more annoying to our neighbors as it is to us. This problem could easily be handled by the purchase of a simple $2. ear piece that plugs into the pager and defeats the external sound, transmitting the message only to the tortured ear of the eager recipients. I believe that there are Radio Shack stores all over the state, so this gadget should be very easy to obtain.

Telephone Courtesy:

How often have any of you had a call put through to you from a fellow physician in the midst of an examination, only to find that he is calling about a specific patient and you first have to obtain a chart before you can carry out an intelligent conversation? There are several very simple basic elements of telephone manners, which if carried out, could ease the burden for all concerned. If you are returning a call to another physician, telling his receptionist that "I am Dr. A, returning Dr. B's call" immediately establishes the precidence of who wants to talk to whom and would expedite matters greatly. The simple manner of you or your receptionist indicating that "this is Dr. A. calling regarding patient C" would also simplify things, enabling her to obtain the patient's chart and bring it into the receiving physician and once more making everyone's lives much simpler.

Community Participation:

Too often do we think that our MD degree gives us enough status and prestige to exempt us from participation in non-medical community activities. Despite my opening remarks, it is apparent that physicians are still opinion formers and influential people in their communities and usually when they speak they are listened to. If organizations such as health planning council, United Way, and other governmental and non-governmental agencies throws a curve ball because they had no physician in-put, we have only ourselves to blame. In our mutual paranoia, we often feel that the deck is stacked against us, so why waste our time? This may be true, but participation never hurts.

I do not feel that any of us, myself included, are absolutely perfect in all of the above areas. An attempt by each and every one of us to improve the public's view of the medical profession and, probably more important, of our individual selves.

The A.M.S.A. Report

ARMA ANNUAL MEETING

DOUG OUTCALT

I would like to share with members of the Arizona Medical Association some thoughts of mine concerning the 1976 convention in Phoenix. As a student delegate from the University of Arizona College of Medicine, I was present and participated in both meetings of the House of Delegates. I was also able to attend the meeting of the Reference Committee on Resolutions as well as some scientific lectures. I would like to congratulate the Arizona Medical Association on the emphasis it places on continuing medical education. Concerning this aspect of the convention I have nothing but praise. Concerning the political process, I come away with mixed emotions.

The big issue of course was malpractice. I was pleased to see the amount of time, money and effort ArMA is spending on this problem. I was impressed by the Malpractice Insurance Crisis Committee and their chairperson, Dr. Jack Brooks. Their approaches were based on a great deal of research and hard work. The discussion that occurred at the reference committee meeting delt with all aspects of the problem except one. There was no mention of the need to increase efforts to police the medical profession and eliminate incompetent physicians. This seemed to me to be a very important omission.

The discussion concerning medicaid at the reference committee was very disappointing. At this meeting ArMA members discussed malpractice for over two hours, medicaid for only 15 minutes. The majority of people present felt that medicaid was not the best thing for the health care of the Arizona people. Regardless, of my disagreement with this viewpoint it was very discouraging to see those members present choose not to speak out on the subject for fear of harming their malpractice efforts.

Another disappointment came with the way resolution 11-76, Decriminalization of sexual practices between consenting adults, was handled at both the Reference Committe and the House of Delegates. This resolution was submitted by Housestaff not with any real hopes of passage but with hopes that productive discussion could take place. It never did. The only "discussion" at the Reference Committe meeting was a series of jokes and quotations from the bible. It was even introduced to the House of Delegates with a joke. After defending the resolution before the House and condeming the attitude with which it has been handled I was gratified with the favorable response I received from a number of the delegates. It gave me hope that the subject can be delt with in the future.

To me the high point of the convention came with the passage by the house of Resolution 12—76 concerning housestaff contracts and collective bargaining. My interest in this as a student and future housestaff members is obvious. The low point came with the tabling of resolution 10—76 concerning support of the 55 MPH speed limit and the decrease in the number of automobile accidents resulting from it.

All things in perspective it was a very interesting and informative convention. I'm sure I was not the only delegate to leave with mixed feelings. I would like to express my appreciation to ArMA for allowing students to attend as delegates and to express viewpoints not only at the convention but in *Arizona Medicine* as well. This is a sign of a healthy organization.

Topics Of Current Medical Interest

BEHAVIORAL HEALTH LIBRARY AND INFORMATION SERVICE

The Arizona Division of Behavioral Health Services announces the initiation of the Behavioral Health Library and Information Service.

The Service is the result of the coordination of the information resources of the Arizona State Hospital Medical Library and the Arizona Drug Abuse Communications Network (DRACON). The following information services are provided to a diversified community-level audience:

Research, Reference, and Dissemination:

1. Articles, books and bibliographies are provided for individuals in the areas of mental health, drug abuse, and alcoholism.
2. Drug Abuse Communications Network (DRACON) — perform hard-data computer searches through the National Clearinghouse for Drug Abuse Information (NCDAI) at Rockville, Maryland.
3. Service Outreach — on-going follow- of previous clientele to appraise them of new information or to suggest assistance.
4. Coordination and delivery of information/communication support services to task forces, workshops and planning activities in the design, implementation and maintenance of Arizona's plan for drug abuse efforts.
5. Publication of a quarterly information bulletin.
6. Distribution of a limited basis of most of the publications of the National Clearinghouse for Drug Abuse Information.

It is important that Arizona addresses itself to the problems surrounding substance abuse and mental illness and commits itself to a coordinated and constant but aggressive action. The Behavioral Health Library and Information Service assumes a vital communication role providing cohesion to that action.

BEHAVIORAL HEALTH INFORMATION SERVICE
Medical Library
2500 East Van Buren Street
Phoenix, Arizona 85008
Phone: 244—1331, Extension 278
JACQUE DOYLE, Medical Librarian
ELLEN HEPNER, DRACON Librarian

DAVID L. BUCKLES NAMED NNHF ADMINISTRATOR

GANADO, ARIZ. — David L. Buckles, former official with an *American Medical Association* program to Vietnam, has been named administrator for the Navajo Nation Health Foundation at Ganado, Ariz.

The foundation is a nonprofit health organization operating historic Sage Memorial Hospital at Ganado and several family health clinics. Established in July, 1974, the foundation is the nation's first Indian-managed health system, with a patient load of more than 30,000 per year. The foundation has been served by an acting administrator for the past year.

Buckles, 43, was project administration officer for the AMA's Vietnam Medical School Project in Saigon for two years. The project provided medical training for Vietnamese at eight teaching hospitals in the Saigon area.

For three years prior to that, Buckles was administrative officer for a joint civilian/military health program affecting 270,000 residents of Binh Thuan Province in Vietnam. In 1970, he received the U.S. State Department's Award for Heroism for his actions during the 1968 Tet offensive.

Most recently, Buckles has been field training coordinator for the University of Southern California School of Medicine, Department of Community Medicine and Public Health. Prior to Vietnam, he served 15 years with the U.S. Air Force medical services and later was business manager and administrative assistant for two Californiz hospitals

In Memoriam

OTTO EMIL UTZINGER, M.D.
1888—1976

"It has been my privelege to know many of the Arizona pioneer physicians. They were persons of indomitable courage, striving to do their duty to uplift the medical profession." Thus said E. Payne Palmer, Sr., M.D. another pioneer Arizona physician, introducing Dr. Utzinger the incoming president of the State Society at the 52nd Annual meeting in Tucson April 30, 1943.

Responding Dr. Utzinger stated, in part, "In Arizona we have a striking example of a segment of medicine which of necessity must be taken care of by the state, that dealing with industrial accidents and diseases." Deploring the encroachment of the Federal government into medical practice he recogonized that, "in certain localities it is necessary to provide facilities for medical care. Such is the case in many of the mining communities in this state, where adequate care could not be obtained unless hospitals were built and physicians supplied by industry. It is not all ideal but so far has been the best solution obtainable." It was during his tenure as President that the state society planned the beginning of an independent Arizona Medical Journal, and fostered the legislation for Blue Cross and Blue Shield. The latter, noble in inception, was later to hang like the proverbial albatross around our collective necks!

Born in Oregon, Dr. Utzinger completed his undergraduate AB at Stanford in 1910, and Garnerod an M.D. from prestigious Johns Hopkins in 1914. Then a surgical two years under the famous surgeon John W. T. Finney at the Union Memorial in Baltimore. Joined the Army for WW I, and after a crash course in brain surgery under Harvey Cushing, on to France with a surgical team assigned to forward collective stations. His victory medal has four "battle clasps" — CHATEAU-THIERRE, SAINT MIHIEL, MEUSE-ARGONNE AND VERDUN.

On his return from WW I he refused teaching offers and came to Arizona as chief surgeon to the Kennecott Copper Company Hospital at Ray, Arizona and served there from 1919—1953 when he retired.

Always a booster for organized medicine he assumed an active part-even from his remote position. Since there was no local county society in Pinal county, on his arrival, he joined the Maricopa County Society—later he organized the Pinal Society. This lead to a not so ammusing sequence. When he moved to Scottsdale, on retirement, he sought transfer of his membership to Maricopa County. But was informed he must go through the regular route of application, review by the Board of Censors, two years on probation and finally full membership. At first affronted, his usual good humor prevaled and a former president of the State Association finally re-entered the Maricopa County Society he originally joined thirty odd years before.

Dr. Utzingers varied interest are exemplified by membership in societies.

- American Medical Association
- Southwestern Medical Association
- Arizona Medical Association and in 1943 elected to the Fifty Year Club
- Fellow, American College of Surgery
- Diplomate, American Board of Surgery and member of founders group

- Chief Surgeon Kennecott Copper Corp., Ray Mines Division 1919—1953
- American Legion
- Paradise Valley Country Club

"Patriotism and professional integrity are not short or frenzied outbursts of emotion but the tranquil and steady dedication of a lifetime."

To Mary, his beloved wife, his three sons, John C., Dr. William G., and Dr. H., the steady dedication of a life time as military surgeon, master surgeon and physician, charished husband and father — Doctor Otto Emil Utzinger — his spirit will ever comfort them.

J.W.K.

Letters to Editor

Dr. John W. Kennedy
Chairman, Publishing Committee
Arizona Medicine

Dear Dr. Kennedy,

Thank you for your letter, dated May 25, 1976.

The funding problems of "Arizona Medicine" are very complex and will require a thorough examination of all the options, in order to solve them.

I believe that we shall continue to amplify and improve the professional sections of "Arizona Medicine", without neglecting the legislative and economic material.

Sincerely yours,
George Lastnick, M.D.

John W. Kennedy, M.D.
Editor, Arizona Medicine

Dear Dr. Kennedy:

This is in response to your editorial, "Arizona Medicine To Be Or Not To Be", which appeared in the June issue of the journal.

I was present at the 1976 meeting of the ArMA House of Delegates at which I expressed my opinion with regard to a re-evaluation of the cost benefit of continuing the publication of the journal. I commented at the time that the use of the word "loss" in the resolution to describe the cost of publishing *Arizona Medicine* betrayed a special kind of bias against the journal on the part of those who favor discontinuation of its publication. I also suggested to the House that it did not seem quite right that when we encounter financial "hard times" and belt tightening has to be done, that we always look to those "unnecessary" expenditures having to do with educational activities.

Let's face it, we, like all other organizations, professional and otherwise, basically function for the benefit of ourselves. I would submit that education of our members is in our own interests. As Dr. Fredell pointed out in his article, "Postgraduate Education", in the June issue of the journal, the day of accountability will soon be upon us. We must show evidence of activities by ArMA that are educational and directed at improving patient care. We cannot give only lip service to that part of our Association's commitment.

We have an opportunity to make *Arizona Medicine* a truly excellent journal. The response to my request for papers for the May issue reflected a definite interest on the part of the faculty of the College of Medicine to publish in the journal. There were many papers rejected because I felt the May issue should be directed at the primary care physician. There were many excellent articles that described results of current research, both "bench" and clinical. With increased publication of such articles we might attract additional subscribers outside of Arizona.

I see no reason why authors cannot be asked to pay $10 or $15 per published page. We might also ask that they purchase a minimum number of reprints.

In summary, I feel we have a good journal that can become an excellent one. I feel that ArMA has an obligation to its members to continue the journal. Finally, I believe there is no reason why we cannot work toward a "New England Journal of the Southwest."

Respectfully,
George D. Comerci, M.D.
Associate Dean, Continuing Medical
Education and Outreach

Bruce Robinson
Managing Editor Arizona Medicine

Dear Mr. Robinson:

Thank you for allowing me the opportunity to reply to Dr. Yudell's letter of 8 June. *(July issue Arizona Medicine, pg 588. Dr. Vosskuhler's original letter, May issue page 394. ed.)* In regard to his first two paragraphs, I feel that it is not necessary to justify the specialty of Radiology. The Radiologist is an expert uninterested third party whose skill is needed for interpretation of medical images. This is recognized in hospital by-laws mandating that x-rays be interpreted by a Radiologist and not by the specialty physician referring the patient to the hospital. This is required by the Joint Accreditation Commission. The American Society of Neuroradiology and the immediate past president of the American College of Radiology, Dr. Robert Wise of Boston, both state that computerized axial tomography is basically a radiologic procedure. It is also a medical imaging method. By either definition this comes within the preview of radiology.

The patient receives the best possible care when referring specialists consult with the Radiologist regarding parameters of this diagnostic study. Optimal patient care is the final end result for which we aim.

The second to last paragraph of my letter mentions two dangers when specialists refer patients to a high priced examination from which they receive a revenue. Self-referral for an expensive procedure very frequently results in over-utilization. The other danger is that of competence. I reiterate that the Radiologist doing these procedures virtually full-time will be more proficient than the Neurologist who does them as a part of the evaluation of some of his patients.

I certainly concur with Dr. Yudell's quotation from the statement of the American Academy of Neurology emphasizing that Neurologists must be involved in decision making in computed tomography. The method is a complex one and study is necessary so that findings can be intellegently discussed. However, I disagree with the second quotation which specifies that only specialists trained in Neurologic disorders are qualified to interpret computed tomography. I feel strongly that the Neuro-radiologist with special training in CT will be better qualified than a Neurologist or a Neuro-surgeon, both in using the examination and in maintaining the necessary quality control.

In conclusion I reiterate the stand taken in my letter to the Editor published in the May issue of *Arizona Medicine*. I feel that optimal patient care will result from referral of patients to Radiologists operating computerized tomography units. I feel that self-referral of patients for this expensive examination involves two very real dangers in the fields of competence and over-utilization.

Yours,
J.W. Vosskuhler, M.D.

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

THE ARIZONA MEDICAL ASSOCIATION, INC.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc. held in the Kudu Room, Safari Hotel, Scottsdale, AZ on Saturday May 1, 1976, a quorum being present, convened at 12:26 p.m., Edward Sattenspiel, M.D., President and Chairman, presiding:

COMMITTEE CONFIRMATION

IT WAS MOVED AND CARRIED TO CONFIRM THE FOLLOWING APPOINTMENTS:
ARTICLES OF INCORPORATION AND BYLAWS COMMITTEE TERM 1976-77

- *Charles E. Henderson, M.D., Chairman
- *William E. Crisp, Jr., M.D., Secretary
- *Philip E. Dew, M.D.
- *Paul B. Jarrett, M.D.
- *John F. Kahle, M.D., President-Elect
- *Edward Sattenspiel, M.D., President
- **William C. Scott, M.D.
- *Clarence E. Yount, Jr., M.D.

*indicates new appointment or reappointment

BENEVOLENT AND LOAN FUND COMMITTEE

	Term
*Arthur V. Dudley, Jr., M.D., Chairman	1976-77
	1976-79
*John T. Clymer, M.D., Treasurer Specified	1976-77

*William E. Crisp, M.D., Secretary	1976-77
**Daniel C. DeBoer, M.D.	1976-79
**William Farr, M.D.	1976-79
R. Lee Foster, M.D.	1974-77
*Andrew M. Goldner, Ph.D.-Assistant Dean-specified	1976-79
*John F. Kahle, M.D., President-Elect	1976-77
Edward J. Lefeber, M.D.	1975-78
*Edward Sattenspiel, M.D., President	1976-77
*Cecil C. Vaughn, Jr., M.D.	1976-79
*Karl E. Voldeng, M.D.	1976-79

FINANCE COMMITTEE

*John T Clymer, M.D., Chairman, Treasurer Specified	1976-77
*Robert F. Crawford, M.D.	1976-79
*William E. Crisp, Jr., M.D., Secretary	1976-77
*Richard L. Dexter, M.D.	1976-79
Herbert G. Erhart, Jr., M.D.	1975-78
*John R. Green, M.D.	1976-79
James L. Grobe, M.D.	1974-77
*John F. Kahle, M.D., President-Elect	1976-77
*Richard T. McDonald, M.D.	1976-79
*Theodore L. Mobley, M.D.	1976-79
*Robert P. Purpura, M.D.	1976-79
*Edward Sattenspiel, M.D., President	1976-77
Seymour I. Shapiro, M.D.	1974-77
Otto S. Shill, Jr., M.D.	1974-77
George Wallace, M.D.	1975-78
*George H. Yard. M.D.	1976-79

GOVERNMENTAL SERVICES COMMITTEE

	Term
*Wallace A. Reed, M.D., Chairman	1976-77 1976-79
*John A. Ash, M.D.	1976-77
**Allan Beigel, M.D.	1976-79
*William E. Crisp, M.D., Secretary	1976-77
Donn G. Duncan, M.D.	1975-78
Walter R. Eicher, M.D.	1975-78
Alan L. Engelberg, M.D.	1974-77
*Lloyd S. Epstein, M.D.	1976-79
*John F. Kahle, M.D., President Elect	1976-77
Charles Kalil, M.D.	1975-78
Louis C. Kossuth, M.D.	1974-77
Joseph J. Likos, M.D.	1974-77
Joseph L. Marcarelli, M.D.	1975-78
*Dermont W. Melick, M.D.	1976-79
Arthur D. Nelson, M.D.	1974-77
Helen M. Roberts, M.D.	1975-78
*Edward Sattenspiel, M.D., President	1976-77
Marvin C. Schneider, M.D.	1974-77

GRIEVANCE COMMITTEE

	Term
William C. Scott, M.D., Chairman, Past-Pres. Specified	1976-77
*Richard W. Abbuhl, M.D.	1976-79
Walter Brazie, M.D.	1975-78
William E. Crisp, M.D., Secretary	1976-77
Philip E. Dew, M.D.	1975-78
Richard E. H. Duisberg, M.D.	1975-78
David Glow, M.D.	1974-77
John B. Jamison, M.D.	1975-78
John F. Kahle, M.D., President-Elect	1976-77
**Philip W. Kantor, M.D.	1976-79
*Richard T. McDonald, M.D.	1976-79
Albert J. Ochsner, M.D.	1974-77
Robert P. Purpura, M.D.	1975-78
Edward Sattenspiel, M.D., President	1976-77

HEALTH MANPOWER COMMITTEE

	Term
Louis C. Kossuth, M.D., Chairman	1976-77/ 1974-77
*Casey D. Blitt, M.D.	1976-77
William E. Crisp, Jr., M.D., Secretary	1976-77
Bruce N. Curtis, M.D.	1974-77
John F. Kahle, M.D., President Elect	1976-77
**John J. Kelley, M.D.	1976-79
Alexander Kelter, M.D.	1975-78
Edward Lefeber, Jr., M.D.	1975-78
Roger A. Lueck, M.D.	1975-78
**Marcus M. Marble, M.D.	1976-79
Dermont W. Melick, M.D.	1974-77
John B. Miller, M.D.	1975-78
Andrew W. Nichols, M.D.	1974-77
**James S. Pucelik, M.D.	1976-79
Robert St. John, M.D.	1975-78
Edward Sattenspiel, M.D., President	1976-77
H. Stephens Thomas, M.D.	1975-78
**Kevin E. Ware, D.O.	1976-79
Herbert L. Winograd, M.D.	1975-78

HISTORY AND OBITUARIES

	Term
John W. Kennedy, M.D., Chairman, Editor-in-Chief Spec.	1976-77
*Francis J. Bean, M.D.	1976-79
*Walter Brazie, M.D.	1976-79
William E. Crisp, M.D., Secretary	1976-77
*G. Bland Giddings, M.D.	1976-79
*John R. Green, M.D.	1976-79
John F. Kahle, M.D., President-Elect	1976-77
Abe I. Podolsky, M.D.	1975-78
Edward Sattenspiel, M.D., President	1976-77
*Jay L. Sitterley, M.D.	1976-79

LEGISLATIVE COMMITTEE

	Term
**Selma Targovnik, M.D., Chairperson	1976-77/ 1976-79
Richard W. Abbuhl, M.D.	1975-78
Harrison G. Butler III, M.D.	1975-78
James E. Campbell, M.D.	1974-77
John S. Carlson, M.D.	1975-78
*W. Scott Chisholm, M.D.	1976-79
*Sam C. Colachis, Jr., M.D.	1976-79
Richard L. Collins, M.D.	1975-78
William E. Crisp, M.D., Secretary	1976-77
**Michael R. Geyser, M.D.	1976-79
Donald F. Griess, M.D.	1975-78
John P. Holbrook, M.D.	1974-77
John F. Kahle, M.D., President-Elect	1976-77
Gerald Marshall, M.D.	1975-78
Richard McGill, D.O.	1975-78
Gerald F. McNally, M.D.	1974-77
Donald R. Miles, M.D.	1974-77
*R. Michael O'Harra, M.D.	1976-79
Ratibor Pantovich, D.O.	1975-78
O. Melvin Phillips, M.D.	1974-77
Wilfred M. Potter, M.D.	1974-77
Edward Sattenspiel, M.D., President	1976-77
*Paul Schnur, M.D.	1976-79

Berton Siegel, D.O.	1975-78
**Jerome H. Targovnik, M.D.	1976-79
John Vosskuhler, M.D.	1975-78
Dennis Weiland, M.D.	1974-77

MATERNAL AND CHILD HEALTH CARE COMMITTEE

	Term
**William C. Scott, M.D., Chairman	1976-77
	1976-79
Frederic W. Baum, M.D.	1975-78
**James F. Blute III, M.D.	1976-79
**Norman B. Brown, M.D.	1976-79
Walter B. Cherny, M.D.	1974-77
C. Donald Christian, M.D.	1975-78
Warren A. Colton, Jr., M.D.	1975-78
William E. Crisp, Jr., M.D., Secretary	1976-77
William J. R. Daily, M.D.	1974-77
Jack H. Demlow, M.D.	1975-78
Glenn M. Friedman, M.D.	1975-78
*Harlan R. Giles, M.D.	1976-79
Raymond J. Jennett, M.D.	1974-77
John F. Kahle, M.D., President Elect	1976-77
Edward Kilgore, M.D.	1975-78
Walter K. Lippard III, M.D.	1975-78
Belton P. Meyer, M.D.	1976-79
**Sister Alice Montgomery	1976-79
*William J. Moore, M.D.	1976-79
William G. Payne, M.D.	1975-78
David Pent, M.D.	1975-78
Hermann S. Rhu, Jr., M.D.	1975-78
Edward Sattenspiel, M.D., President	1976-77
Paul A. Whitmore, D.O.	1974-77

MEDICAL ECONOMICS

	Term
*Robert P. Purpura, M.D., Chairman	1976-77
	1975-78
Richard S. Armstrong, M.D.	1975-78
*Albert C. Asendorf, M.D.	1976-79
Chester G. Bennett, M.D.	1974-77
Avi Ben-Ora, M.D.	1974-77
Sam C. Colachis, Jr., M.D.	1974-77
William E. Crisp, Jr., M.D., Secretary	1976-77
*Charles F. Dalton, M.D.	1976-79
George L Hoffman, M.D.	1974-77
Frederick W. Jensen, Jr., M.D.	1975-78
John F. Kahle, M.D., President-Elect	1975-78
Howard N. Kandell, M.D.	1974-77
Patrick P. Moraca, M.D.	1974-77
Edward Sattenspiel, M.D., President	1976-77
*Richard W. Switzer, M.D.	1976-79
**Martin L. Welch, M.D.	1976-79
Burton E. Weissman, M.D.	1974-77
Reginald J. Zeluff, M.D.	1975-78

MEDICAL EDUCATION COMMITTEE

	Term
*Robert E. T. Stark, M.D., Chairman	1976-77
	1975-78
James E. Brady, M.D.	1975-78
*Daniel B. Carroll, M.D.	1976-79
Melvin L. Cohen, M.D.	1976-79
George D. Comerci, M.D.	1975-78
William E. Crisp, M.D., Secretary	1976-77
*David J. Crosby, M.D.	1976-79
*Kenneth A. Dregseth, M.D.	1976-79
Francis T. Flood, M.D.	1975-78
*Harry W. Hale, Jr., M.D.	1976-79
*Robert E. Hastings, M.D.	1976-79
**John W. Heaton, M.D.	1976-79
M. Wayne Heine, M.D.	1975-78
John F. Kahle, M.D., President-Elect	1976-77
*Howard N. Kandell, M.D.	1976-79
Jack M. Layton, M.D.	1974-77
Albert Rizzoli, M.D.	1975-78
Edward Sattenspiel, M.D., President	1976-77
William F. Sheeley, M.D.	1974-77
*John J. Standifer, M.D.	1976-79
Lawrence Z. Stern, M.D.	1975-78
**David C. H. Sun, M.D.	1976-79
Ashton B. Taylor, M.D.	1975-78
**Charles A. Trahern, M.D.	1976-77
Albert G. Wagner, M.D.	1975-78

PHYSICIAN REHABILITATION COMMITTEE

	Term
*Richard E. H. Duisberg, M.D., Chairman	1976-77
	1975-78
Otto L. Bendheim, M.D.	1975-77
William E. Bishop, M.D.	1975-77
John T. Clymer, M.D.	1975-78
William E. Crisp, M.D., Secretary	1976-77
**Everett W. Czerny, M.D.	1976-79
Donald L. Damstra, M.D.	1975-78
**Wesley E. Green, M.D.	1976-79
John F. Kahle, M.D., President-Elect	1976-77
Laurence M. Linkner, M.D.	1975-78
Edward Sattenspiel, M.D., President	1976-77
Stephen C. Scheiber, M.D.	1976-79
*Walter B. Tomlinson, M.D.	1976-79
Karl E. Voldeng, M.D.	1975-77
Eleanor A. Waskow, M.D.	1975-77

PROFESSIONAL COMMITTEE

	Term
*Joseph W. Hanss, Jr., M.D., Chairman	1976-77
	1975-78
Paul M. Bindelgas, M.D.	1974-77
Paul B. Borgeson, M.D.	1974-77
John A. Bruner, M.D.	1975-78
L. Philip Carter, M.D.	1975-78
William E. Crisp, M.D., Secretary	1976-77
**Alan L. Gordon, M.D.	1976-79
James L. Grobe, M.D.	1974-77
James M. Hurley, M.D.	1974-77
*Helen Johnson, M.D.	1976-79
John F. Kahle, M.D., President-Elect	1976-77
**James G. Lawson, M.D.	1976-79
*Laurence M. Linkner, M.D.	1976-79
*Joseph H. Reno, M.D.	1976-79
Edward Sattenspiel, M.D., President	1976-77
*Donald F. Schaller, M.D.	1976-79
George A. Spendlove, M.D.	1974-77
Neil O. Ward, M.D.	1975-78

PUBLIC RELATIONS COMMITTEE

	Term
**Charles H. Finney, M.D., Chairman	1976-77
	1976-79
W. David Ben-Asher, M.D.	1975-78
**James R. Callison, M.D.	1976-79
**William G. Chaffee, Jr., M.D.	1976-79
Ronald L. Christ, M.D.	1975-78
William E. Crisp, M.D., Secretary	1976-77

Julian DeVries, Medical Editor, AZ Republic	1975-78
**Theodore G. Dodenhoff, M.D.	1976-79
**Merlin K. DuVal, M.D.	1976-79
**Ira B. Ehrlich, M.D.	1976-79
Edward B. Grothaus, M.D.	1974-77
John F. Kahle, M.D., President-Elect	1976-77
**Gennaro Licosati, M.D.	1976-79
**Mrs. Nikki Mertz	1976-79
Edward Sattenspiel, M.D., President	1976-77
Morton S. Thomas III, M.D.	1974-77
**Alan Yudell, M.D.	1976-79

PUBLISHING COMMITTEE

	Term
John W. Kennedy, M.D., Chairman, Editor-Specified	1976-77
William E. Crisp, Jr., M.D., Secretary	1976-77
Walter V. Edwards, M.D.	1974-77
**Warren F. Gorman, M.D.	1976-79
**John R. Green, M.D.	1976-79
John F. Kahle, M.D., President-Elect	1976-77
Gerald Kaplan, M.D.	1974-77
George Lastnick, M.D.	1975-78
William B. McGrath, M.D.	1975-78
Stanley H. Nordmo, M.D.	1975-78
Edward Sattenspiel, M.D., President	1976-77
Michael M. Shreiber, M.D.	1974-77
**Murray Spotnitz, M.D.	1976-79
**Donald J. Ziehm, M.D.	1976-79

SCIENTIFIC ASSEMBLY COMMITTEE

	Term
*Luis S. Tan, M.D., Chairman	1976-77
	1974-77
Merrill M. Abeshaus, M.D.	1975-78
*Suresh C. Anand, M.D.	1976-79
Floyd K. Berk, M.D.	1974-77
*Thomas E. Bittker, M.D.	1976-79
*W. Scott Chisholm, M.D.	1976-79
**George D. Comerci, M.D.	1976-79
William E. Crisp, M.D., Secretary	1976-77
**Boyden Crouch, M.D.	1976-77
Otto Gambacorta, M.D.	1974-77
Jerome Gerendasy, M.D.	1975-78
Laurence M. Haas, M.D.	1975-78
Timothy R. Harrington, M.D.	1975-78
Clifford J. Harris, M.D.	1974-77
Thomas F. Hartley, M.D.	1975-78
Wayne M. Heine, M.D.,	1975-78
Thomas S. Henry, M.D.	1975-78
**Stuart I. Holtzman, M.D.	1976-79
**Efren N. Hufana, M.D.	1976-79
James M. Hurley, M.D.	1974-77
John F. Kahle, M.D., President-Elect	1976-77
Norman N. Komar, M.D.	1976-79
Eugene Leibsohn, M.D.	1974-77
Philip Levy, M.D.	1974-77
**Peter Mao, M.D.	1976-79
William S. Nevin, M.D.	1975-78
Neopito L. Robles, M.D.	1974-77
Edward Sattenspiel, M.D., President	1976-77
**Marvin C. Schneider, M.D.	1976-79
Richard A. Silver, M.D.	1974-77
Donald P. Speer, M.D.	1975-78
Wilbur C. Voss, M.D.	1974-77
Donald J. Ziehm, M.D.	1974-77

ArMPAC BOARD OF DIRECTORS — TERM 1976-77

*Don V. Langston, M.D., Chairman
*Ruben Acosta, M.D.
*James A. Austin, M.D.
*Casey D. Blitt, M.D.
*Walter Brazie, M.D.
*Richard E. Brown, M.D.
*William E. Crisp, Jr., M.D., Secretary
*Arthur V. Dudley, M.D.
*Michael R. Geyser, M.D.
*Timothy R. Harrington, M.D.
*Mrs. Charles E. Henderson
*Stuart I. Holtzman, M.D.
*Mrs. Clare W. Johnson
*John F. Kahle, M.D., President-Elect
*William S. Nevin, M.D.
*Rosamond C. Romero, M.D.
*Edward Sattenspiel, M.D.
*Mrs. Thomas A. Taber
Selma E. Targovnik, M.D.
***John W. Vosshuhler, M.D.**

OTHER BUSINESS

Medicaid Provider Agreement

It was recommended that the Governmental Services Committee review the subject form and prepare a recommendation to the membership as to how they should respond.

Finance Committee

IT WAS MOVED AND CARRIED THAT THE FINANCE COMMITTEE LOOK INTO THE USE OF "ZERO BASED" BUDGETING FOR THE ASSOCIATION'S 1978 ANNUAL BUDGET.

Membership Poll

It was recommended that the proposed poll regarding unified membership and *Arizona Medicine* be brought to the Board of Directors before it is sent to the membership.

Meeting adjourned 1:34 p.m.

William E. Crisp, M.D.
Secretary

ArMA
Medical History

CAMELBACK HOSPITAL
PHOENIX, ARIZONA

History

1952—Catalina Sanitarium (6 = 2-3 bed rooms) —owned by B. Harrison. Toward the end of 1953, the Executive Secretary of the Maricopa County Medical Society pointed out the need for a psychiatric hospital in the Phoenix area. Not a single hospital bed (outside of the antiquated, overcrowded and under-staffed State Hospital) was available for mental patients, who left the state by the hundreds yearly for treatment in California, Texas, even as far away as Kansas and Maryland.

The psychiatrists recognized the merit of this idea, and with their consent and approval a guest ranch, which had been used for a short time as a rest home, was purchased by Otto L. Bendheim, M.D.

A hospital license was obtained from the State Health Department, under whose supervision the hospital has operated since that time.

Since the influx of psychiatrists and patients has exceeded all expectations, new construction of large additions has been necessary, so that the original buildings today comprise only about one-tenth of the total. In other words, approximately 90% of the present building complex has been planned and constructed to our specifications, for our purposes, and by our architects.

Description of Property

The hospital is located in a ten acre citrus grove with mature orange, grapefruit, lemon, and tangerine trees, date palms, royal palms, etc.

The hospital buildings, in California mission architecture with red tile roof, consist of an elongated complex of wings. To the northwest (Ramada Wing) is a closed section for seriously ill patients; to the north an area (soon to be remodelled) for our geriatric patients; the center consists of a beauty shop, lounge, dining room, kitchen, pharmacy, and record room; to the south and east is a wing of private and semi-private rooms, a completely open section (South Wing); to the east a semi-open section (Patio Wing) which presently houses our young adult patients.

Most of the open wing was constructed in 1955 (South Wing), with additional construction in 1960 to provide a new nursing station and 3 additional semi-private rooms for the South Wing, and 10 additional semi-private rooms to make up our Patio Wing. What is now the North Wing, housing our geriatric patients, was added in 1956.

In 1953 a professional building to the west of the hospital, with seven office suites, was constructed.

A second larger professional building was constructed in 1959-1960 and opened February 15, 1960, to provide suites for additional psychiatrists, a clinical laboratory, xray laboratory and electroencephalograph laboratory (the latter three areas are now used as file areas for the psychiatrists). This building also contained a conference room where staff meetings and seminars were held, but this area is now to be remodelled into additional offices.

The two professional buildings were constructed in contemporary style with underground air conditioning.

In 1959, a beautiful landscaped swimming pool was constructed, and additional parking facilities provided.

In 1961, a large new hospital kitchen with entirely new equipment was constructed to serve a capacity of 150 persons per meal.

In March of 1970, an acute wing (Ramada Wing), with Treatment and Recovery Rooms was added. Also an Administration Wing with Offices, and an Adjunctive Therapy Work Shop. Also Xray and Electroencephalographic Laboratories.

In April of 1970, the Patient's Dining Area and Lounge were remodeled with a Snack Bar being added. Also the Kitchen was remodelled with new equipment being purchased. A new Conference Room, covering the entire second floor of this area, is now available.

A. Hospital	No. of Beds	
The Ramada Wing consists of:		
3 Security Rooms	(3)	
4 Private Rooms	4	
6 Semi Private Rooms	12	
2 Wards (4 beds each)	8	
		24
The North Wing consists of:		
4 Private Rooms	4	
4 Semi-Private Rooms	8	
		12
The Patio Wing consists of:		
10 Semi-Private Rooms	20	
		20
The South Wing Consists of:		
7 Private Rooms	7	
13 Semi-Private Rooms	26	
		33
TOTAL BEDS		89

B. Professional Building

The following psychiatrists are tenants of the Professional Building:

	Staff Member Since	Specialty
Eugene Almer, M.D.	1966	Psychiatry
Otto L. Bendheim, M.D.	1954	Psychiatry
Paul M. Bindelglas, M.D.	1961	Psychiatry
James Campbell, M.D.	1971	Psychiatry
Jack Games, M.D.	1972	Psychiatry
Thomas F. Kruchek, M.D.	1962	Psychiatry
Harris Murley, M.D.	1971	Child Psy.
Robert Ranucci, M.D.	1975	Psychiatry
George Saravia, M.D.	1964	Psychiatry
Jerome Szymanski, M.D.	1967	Psychiatry
Floyd Templeton, M.D.	1969	Psychiatry
Mark Wellek, M.D.	1972	Psychiatry
Harold E. McNeely, Ph.D.	1958	Psychology

Bed Occupancy

Average Patients Per Day:

Year Ending		
	1955	18 (approximately)
	1956	23 (approximately)
October 31,	1957	32.4 (exactly)
October 31,	1957	32.4 (exactly)
October 31,	1959	42.8 (exactly)
October 31,	1960	46.3 (exactly)
October 31,	1961	54.7 (exactly)
October 31,	1962	61.3 (exactly)
December 31,	1963	70.6 (exactly)
December 31,	1964	65.5 (exactly)
December 31,	1965	66.2 (exactly)
December 31,	1966	73.8 (exactly)
December 31,	1967	72.5 (exactly)
December 31,	1968	77.6 (exactly)
December 31,	1969	70.9 (exactly)
December 31,	1970	84.4 (exactly)
December 31,	1971	91.3 (exactly)
December 31,	1972	78.3 (exactly)
December 31,	1973	81.94 (exactly)
December 31,	1974	83.98 (exactly)

In July of 1966, Camelback Hospital applied to the Department of Health, Education, and Welfare Health Services and Mental Health Administration of the USPHS for a staffing grant for a Community Mental Health Center, the first such center to be established in Arizona.

A grant in the amount of:
$296,655 was awarded the first year (1967),
$251, 672 was awarded for the second year (1968),
$190,744 was awarded for the third year (1969),
(For 1970, 1971, and 1972 no new monies were awarded under this grant; however, unused monies from previous years were available).
$163,825 was awarded the seventh year (1973)
$152,665 was awarded the eighth year (1974).

Detailed information covering the programs and activities of the Center will be found elsewhere.

Public and Community Relations

Since the Maricopa County Medical Society (membership ca. 900) was instrumental in creating Camelback Hospital, and since our Staff includes members of the Society who are active in its committees, our relations with the non-psychiatric medical profession have been excellent.

Over the years, Camelback Hospital has sponsored:

Occasional breakfasts and luncheons (with guest speakers) for medical fraternal organizations.

Dinner meetings (with speakers on psychiatric subjects) for the USPH Indian Hospitals in Arizona, so the young physicians assigned to these hospitals have an opportunity for a psychiatric orientation.

Other Activities

A. In 1958, we appeared before the Arizona Legislature with the suggestion to change the Commitment Law. As a result, patients can now be committed by Court directly to

Camelback Hospital, making it possible for certified patients to be treated at Camelback Hospital rather than the State Hospital, if they so wish.

B. In 1959, a contractural relationship with the U.S. Public Health Service was established, enabling Indian patients to be treated at Camelback Hospital. Also a similar agreement was reached with the Veterans Hospital to provide care for veterans, when accomodations were not available at any of the three V.A. Hospitals in Arizona.

C. The administrator of Camelback Hospital represented the Arizona Hospital Association in Washington, D.C. in May of 1965, where he attended the meeting of the Senate Finance Committee and spoke with Arizona senators regarding changes in the Medicare Bill to include specialty hospitals.

D. Over the years, we have been in constant contact with the large industrial and mining corporations and labor unions to make available psychiatric hospital benefits for their employees and dependents.

E. Camelback Hospital has worked with the Arizona Insurance Commission concerning misleading language contained in some policies, which purport to cover hospitalization for mental illness and then define hospitals as institutions having emergency room, O.B., and surgical facilities. Currently we follow-up directly with insurance carriers as to reasons for non-coverage of hospitalization at this facility.

F. Camelback Hospital is a participating hospital in the Health Insurance Program (Medicare), also CHAMPUS. Other agencies utilizing the facilities and services of Camelback Hospital are Industrial Commission of Arizona, State Institution for Juveniles, and formerly Job Corps, Peace Corps, and NARA.

G. There has been active participation by our Staff in community projects and organizations such as the Arizona Mental Health Association, State and County Medical Societies, WICHE, Intermountain Psychiatric Association, Academy for Religion and Mental Health, LEAP, Arizona Town Hall, Job Corps, Peace Corps, Arizona Hospital Association, Health Facilities Planning Council, CODAC, etc.

H. The Medical Director of Camelback Hospital appeared before the Senate Subcommittee in September of 1972 in regard to the Navajo-Hopi Territorial Dispute.

I. The Hospital and its Administrator are members of:
   The Arizona Hospital Association (President, 1967-1968)
   The American Hospital Association (Chairman, Governing Council, Psychiatric Hospital Section, 1969-1970)
   Association of Private Psychiatric Hospitals (Vice President, '66-67)
   Association of Western Hospitals
   American College of Hospital Administrators
   Arizona Mental Health Association.

J. In April of 1970, the Citizen's Advisory Board of Camelback Hospital Mental Health Center was established.

K. In April of 1973, a lease agreement was made with the Scottsdale Methodist Church to be used for our Day Care Program. This program has since been re-located at Camelback Hospital.

L. In January 1974, Stuart M. Gould, Jr., M.D. was appointed as full-time Medical Director of Camelback Hospital.

M. In March, 1974, the Camelback Hospital Western Institute of Psychodrama was established with Mrs. Elaine Goldman as Director.

N. In July of 1974, Camelback Hospital initiated an Employee Health Program. Karl Voldeng, M.D., a member of our Consulting Medical Staff since 1963 is our Personnel Physician.

Corporate Structure

1. From 1954-1967, the land and buildings were owned by the Phoenix Institute of Neurology and Psychiatry, an Arizona Corporation, and these facilities were leased by Camelback Hospital, an Arizona non-profit corporation.
2. In 1959, a non-profit corporation, the Arizona Foundation for Neurology and Psychiatry was established mainly for research and educational purposes.
3. In 1962, the Corporation purchased Camelback Hospital, and has operated it since that time as a non-profit community corporation.
4. In 1967, the Foundation purchased the buildings and grounds from the Phoenix Institute of Neurology and Psychiatry, and became owner and operator of Camelback Hospital and the Professional Building.
5. In March of 1972, the name of the Arizona Foundation for Neurology and Psychiatry was legally changed to Camelback Hospital, Incorporated, and the Mental Health Center became the Camelback Hospital Mental Health Center.
6. In April of 1973, Camelback Hospital, Incorporated purchased the building owned by the Arizona Mental Health Association and which housed our Camelback Hospital Mental Health Out-patient Department.

ARIZONA MEDICINE

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CONTRIBUTIONS

The Editor sincerely solicits contributions of scientific articles for publication ARIZONA MEDICINE. All such contributions are greatly appreciated All will be given equal consideration.

Material submitted for publication in ARIZONA MEDICINE should conform to the following policies:

1. Manuscripts, including references or bibliography, should be typewritten, double-spaced, on one side of the paper only, and the original and a carbon enclosed

2. Be guided by the general rules of medical writing as followed by the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

3. Although the Editors try to catch inaccuracies, the ultimate responsibility is the author's.

4. Articles are accepted for publication only if they are contributed exclusively to this Journal Ordinarily, contributors will be notified within 60 days if a manuscript is accepted for publication. Every effort will be made to return unused manuscripts.

5. The Journal reserves the right to edit all material.

6. Reprints will be supplied to the author at printing cost.

Future Medical Meetings

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DESERT SAMARITAN HOSPITAL, MESA
GOOD SAMARITAN HOSPITAL, PHOENIX
PHOENIX INDIAN MEDICAL CENTER
MARICOPA COUNTY GENERAL HOSPITAL, PHOENIX
MEMORIAL HOSPITAL, PHOENIX
ST. LUKE'S HOSPITAL AND MEDICAL CENTER, PHOENIX
ST. JOSEPH'S HOSPITAL AND MEDICAL CENTER, PHOENIX
TUCSON HOSPITALS MEDICAL EDUCATION PROGRAM, TUCSON
VETERANS ADMINISTRATION CENTER, PRESCOTT

CONTINUING MEDICAL EDUCATION ACTIVITIES SPONSORED BY THESE INSTITUTIONS RECEIVE CATEGORY 1 CREDIT FOR THE ArMA CERTIFICATE IN CONTINUING MEDICAL EDUCATION AND THE AMA PHYSICIAN'S RECOGNITION AWARD.

A HEALTH DELIVERY SEMINAR

Mountain Shadows, Scottsdale, AZ
September 10, 1976

SPONSOR: Arizona Regional Medical Program

CONTACT:
Dermont W Melick, M.D.
Arizona Regional Medical Program
608 N. Jefferson Ave.
Tucson AZ 85711

Approved for Continuing Medical Education Hours toward the ArMA Certificate.

INFECTION CONTROL SEMINAR

October 6 & 7, 1976
Hilton Inn, Tucson, AZ

SPONSOR: Tucson Inter-Hospital Infection Control Committee, & CME Office, U of A College of Medicine

CONTACT:
George D. Comerci, M.D.
U of A College of Medicine
Tucson, AZ 85724

Aproved for 8 hours toward the ArMA Certificate in Continuing Medical Education.

MANAGEMENT OF THE MULTI-PROBLEM ELDERLY PATIENT

October 8 - 9, 1976
Del Webb Townehouse, Phoenix, AZ

SPONSOR: American Geriatrics Society & U of A College of Medicine

CONTACT:
Julius Schwimmer, M.D., FACC
13000 N. 103rd Ave.
Sun City, AZ 85351

Approved for 14 required hours toward the ArMA Certificate in Continuing Medical Education

DISTRICT VIII MEETING OF THE AMERICAN COLLEGE OF OBSTETRICIANS & GYNECOLOGISTS

October 10-14, 1976
The Adams Hotel, Phoenix, AZ

SPONSOR: District VIII, American College of Obstetricians & Gynecologists

CONTACT:
David Pent, M.D.
2021 N. Central
Phoenix, AZ 85004

Approved for 27 required hours toward the ArMA Certificate in Continuing Medical Education.

GLENDALE SAMARITAN HOSPITAL MEDICAL EDUCATION LECTURES

January through December, 1976, 7 a.m.

SPONSOR: Glendale Samaritan Hospital

CONTACT:
Gerald Marshall, M.D.
6010 W. Northern
Glendale, AZ 85301

Approved for 5 required hours toward the ArMA Certificate in Continuing Medical Education.

STAFF EDUCATION CONFERENCE

Wednesdays, Weekly, 1 p.m.
Arizona State Hospital, Phoenix, AZ

SPONSOR: Arizona State Hospital

CONTACT:
Howard E. Wulsin, M.D.
Arizona State Hospital
2500 E. Van Buren
Phoenix, AZ 85008

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PSYCHIATRIC GRAND ROUNDS

Every Wednesday, September - May
4:00 p.m. to 5:30 p.m.
Arizona Medical Center, Tucson, AZ, Rm. 8403

SPONSOR: Department of Psychiatry, U of A College of Medicine

CONTACT:
Roy N. Killingsworth, M.D.
Dept. of Psychiatry
Arizona Medical Center
Tucson, AZ 85724

Approved for 1½ per session toward the ArMA Certificate in Continuing Medical Education.

MONTHLY MEETING OF TUCSON RADIOLOGISTS

Last Tues. of Month
Plaza International, Tucson, AZ

SPONSOR: U of A Medical Center, Dept. of Radiology

CONTACT: Irwin M. Freundlich, M.D.
Arizona Medical Center
Dept. of Radiology
Tucson, AZ 85724

Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

GRAND ROUNDS

Each Thursday 7 a.m.-8 a.m.
St. Mary's Hospital and Health Center,

SPONSOR: Depts. of Medicine, Surgery, Radiology, Pathology and Family Practice.

CONTACT:
Richard Silver, M.D.
Chairman, Medical Education
and Library Committee
Century Medical Plaza, Suite 160
1701 West St. Mary's Road
Tucson, AZ 85703

Approved for 1 required hour per round toward the ArMA Certificate in Continuing Medical Education.

BI-MONTHLY MEDICAL EDUCATION SEMINAR

Every other Wed. AM
Begin 7/3/74
Maryvale Samaritan Hospital, Phoenix, AZ

SPONSOR: Medical Staff Maryvale Hospital

CONTACT:
Thomas J. Groves, M.D.
6037 W. Elm St.
Phoenix, AZ 85033

Approved for 1 required hour per session toward the ArMA Certificate in Continuiing Medical Education.

TUMOR BOARD CONFERENCE

Every Friday at Noon
Kiva Conference Room
Phoenix Memorial Hospital

SPONSOR: Phoenix Memorial Hospital

CONTACT:
Howard Kimball, M.D.
333 West Thomas Road
Phoenix, AZ 85013

Approved for credit toward the ArMA Certificate in Continuing Medical Education.

MONTHLY MEDICAL EDUCATION SEMINAR

Third Monday of the Month
Kiva Conference Room
Phoenix Memorial Hospital

SPONSOR: Medical Staff of Memorial Hospital

CONTACT:
George Scharf, M.D.
1201 South 7th Avenue
Phoenix, AZ 85007

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PULMONARY DISEASE GRAND ROUNDS

Mondays — 12 Noon
D-5 North Conference Rm., Good Samaritan Hospital, Phoenix, AZ

SPONSOR: Pulmonary Disease Teaching Service Good Samaritan Hospital

CONTACT:
Bernard E. Levine, M.D.
Pulmonary Function Laboratory
Good Samaritan Hospital
1033 E. McDowell Hospital
Phoenix, AZ 85006

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

CAMELBACK HOSPITAL CLINICAL CONFERENCE

3rd Tuesday monthly
Camelback Hospital, Phoenix, AZ

SPONSOR: Camelback Hospital

CONTACT:
Stuart M. Gould, Jr., M.D.
Medical Director
Camelback Hospital
5055 N. 34th St.
Phoenix, AZ 85018

Approved for 1 elective hour per session toward the ArMA Certificate in Continuing Medical Education.

PATIENT STAFFING CONFERENCE

Three times weekly
Camelback Hospital, Phoenix, AZ

SPONSOR: Camelback Hospital

CONTACT:
Stuart M. Gould, Jr., M.D.
Medical Director
Camelback Hospital
5055 N. 34th St.
Phoenix, AZ 85018

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

MORBIDITY & MORTALITY CONFERENCE

2nd Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Dir. Medical Education
7300 E. 4th St.
Scottsdale, AZ

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CLINICAL PATHOLOGICAL CONFERENCE

4th Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Director of Medical Education
7300 E. 4th St.
Scottsdale, AZ

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

MEDICAL GRAND ROUNDS

3rd Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Dir. of Medical Education
7300 E. 4th St.
Scottsdale, AZ

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

FAMILY PRACTICE CONFERENCE

1st Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Dir. of Medical Education
7300 E. 4th St.
Scottsdale, AZ

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CLINICAL CANCER CONFERENCE

3rd Wednesday Every Month
Butler Bldg. Conference Room
Good Samaritan Hospital, Phoenix, AZ

SPONSOR: Good Samaritan Hospital

CONTACT:
John A. Bruner, M.D.
926 E. McDowell Road
Phoenix, AZ 85006

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

SURGICAL GRAND ROUNDS
4TH TUESDAY OF EACH MONTH

Hospital Auditorium
Baptist Hospital, Phoenix

SPONSOR: Baptist Hospital Phoenix

CONTACT·
James B. Shields, M.D.
6036 N. 19th Ave.
Phoenix, AZ 85015

Approved for 1½ required hours per month toward the ArMA Certificate in Continuing Medical Education.

CARDIOLOGY CONFERENCE

Weekly — Friday 8-9 a.m.
St. Mary's Hospital Auditorium
Tucson, AZ

SPONSOR: St. Mary's Hospital

CONTACT:
A. L. Forte, M.D.
St. Mary's Hospital
Tucson, AZ 85724

Approved for one required hour toward the ArMA Certificate in Continuing Medical Education.

PROBLEM CASE WORKSHOPS

3rd Monday of each month 7:30 a.m.
Room 4410, Arizona Medical Center, Tucson, AZ

SPONSOR: Division of Ophthalmology, U of A
College of Medicine

CONTACT:
H. E. Cross, M.D., Ph.D.
Arizona Medical Center
Dept. of Surgery
Tucson, AZ

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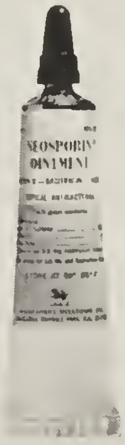
Prophylactically, the ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing **CONTRAINDICATIONS:** Not for use in the eyes or external ear canal if the eardrum is perforated This product is contraindicated in those individuals who have shown hypersensitivity to any of the components **WARNING:** Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended **PRECAUTIONS:** As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi Appropriate measures should be taken if this occurs. **ADVERSE REACTIONS:** Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin Ototoxicity and nephrotoxicity have been reported (see Warning section)

Complete literature available on request from Professional Services Dept PML



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FOR FAMILY AND GENERAL PRACTITIONERS, GENERAL INTERNISTS AND GENERAL PEDIATRICIANS

Designed as a review of the principles and procedures involved in the management of patients with problems commonly encountered in primary care, the program includes a morning lecture series and afternoon elective problem-solving sessions, seminars, and demonstrations. Tuition for the course is $250 with registration required no later than October 8.

General lectures will cover the following topics: scintigraphy, body scanning-computerized axial tomography, ultrasound, prevention of heart attack, management of hypertension, management of angina pectoris, pre-CCU management of heart attack, office management of arrhythmias, management of osteoarthritis and rheumatoid arthritis, headache, depression, transient ischemic attacks, management of common cutaneous complaints, management of urinary tract infections.

Elective sessions will include: **family dynamics:** crisis points in family life, problems in school, problems in middle life, problems surrounding death; **problems related to athletic activity:** the upper extremity, the lower extremity, the spine, reconditioning in middle life; **acid-base problems:** general principles, acidosis, alkalosis, mixed problems; **problem-oriented records:** general principles, the defined data base, construction of the problem list, workshop; **water and electrolyte problems:** water, sodium, potassium, miscellaneous problems; **immunology:** cellular basis of immunity, the immunoglobulins, antigen-antibody complex disease, allergy.

Faculty for this course will consist of twenty-five physicians of the Stanford University School of Medicine.

INTENSIVE CARE NOVEMBER 1-5, 1976

FOR ALL PHYSICIANS INVOLVED IN THE CARE OF THE CRITICALLY ILL OR INJURED PATIENT

This course will review in detail the current status of management principles and procedures applicable to critically ill and critically injured persons. Topics of general interest will be covered in morning lectures, with the afternoon program offering a choice of problem-solving sessions and seminars and demonstrations in specialized topics. Twenty-nine Stanford University School of Medicine faculty members will participate.

General lectures will cover: hemodynamic monitoring; antiarrhythmic drugs; surgery for ischemic heart disease; respiratory failure: pathophysiology, manifestations and management, the principles and practical use of ventilators; hemostatic mechanisms; common bleeding problems: congenital-acquired; diabetic ketoacidosis and hyperosmolar coma; lactic acidosis; coma; thromboembolism; adult respiratory distress syndrome.

Elective sessions will include: **EKG problems:** acute myocardial infarction, tachyarrhythmias, bradyarrhythmias, ST-T changes in severe illness; **blood gas and acid-base problems:** respiratory acidosis and alkalosis, metabolic acidosis, metabolic alkalosis, oxygen transport; **water and electrolyte problems:** water, sodium, potassium, miscellaneous problems; **acute myocardial infarction:** cardiogenic shock, arrhythmias, emergency surgery, acute rehabilitation in CCU; **ICU methods:** resuscitation, monitoring: CVP and arterial lines, intracardiac and pulmonary lines, pacemakers; **trauma:** injuries to the face, the hands, the chest, the head and spine; **neonatal crises:** neonatal asphyxia, respiratory distress syndrome, sepsis, metabolic crises in the newborn; **ICU management problems:** use of blood components, acute renal failure, antibiotics in septic crises, hyperalimentation.

Tuition for this course is $250 with registration required no later than October 29. Early registration is advisable as this course has been oversubscribed in previous years.

FOR COMPLETE DETAILS SEND COUPON

Clip and mail to: OFFICE OF POSTGRADUATE MEDICAL EDUCATION
Stanford University School of Medicine, Room TA145, Stanford, California 94305
Telephone (415) 497-5594

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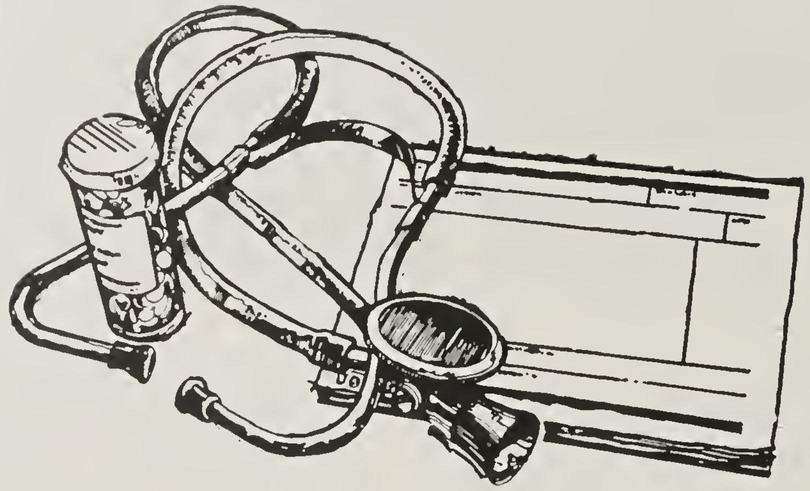

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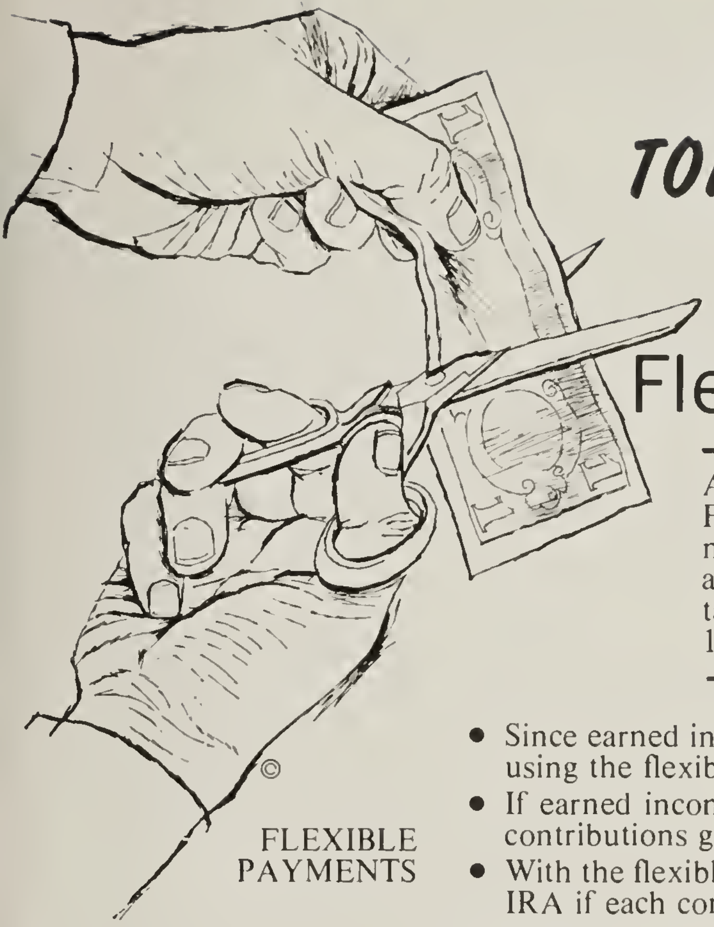

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Your dollars will accumulate on a guaranteed growth basis. In addition, we are currently paying a non-guaranteed, higher interest rate than the guaranteed amount. The following illustrates both the guaranteed and current non-guaranteed accumulation and income at age 65.

Assume $100 monthly contributions to age 65
Monthly Benefit at 65 for 120 months certain and life

Age at Issue	Guaranteed Dollar Accumulation	Current (Non-Guar.) Accumulation	Monthly Benefit Guaranteed Male	Monthly Benefit Guaranteed Female	Monthly Benefit Non-Guaranteed Male	Monthly Benefit Non-Guaranteed Female
35	$58,458	$102,476	$364	$330	$818	$764
45	32,164	45,264	205	186	361	337
55	13,523	15,492	86	78	123	115

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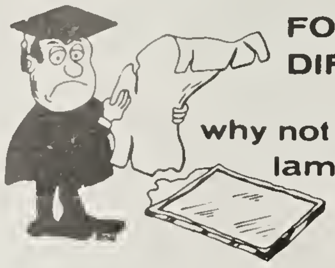

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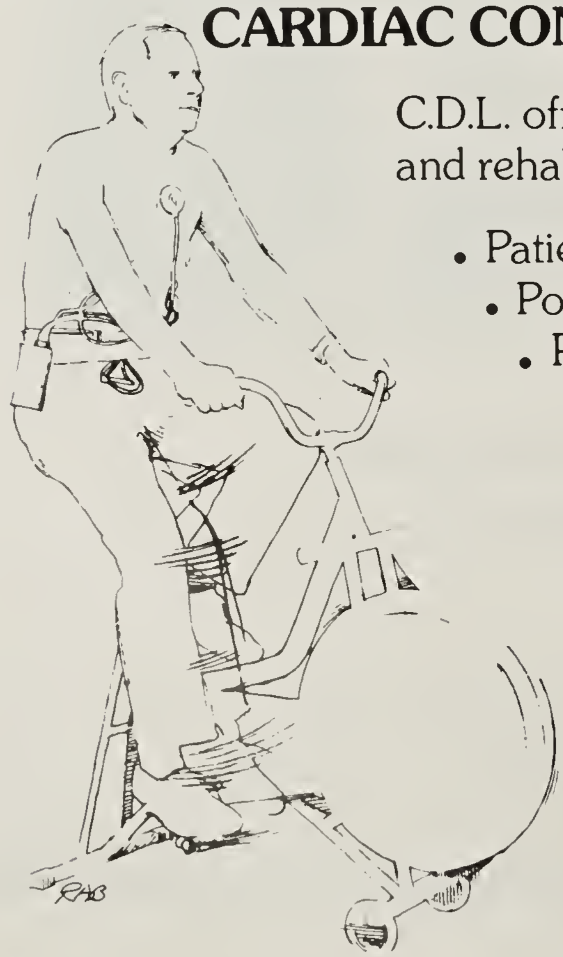

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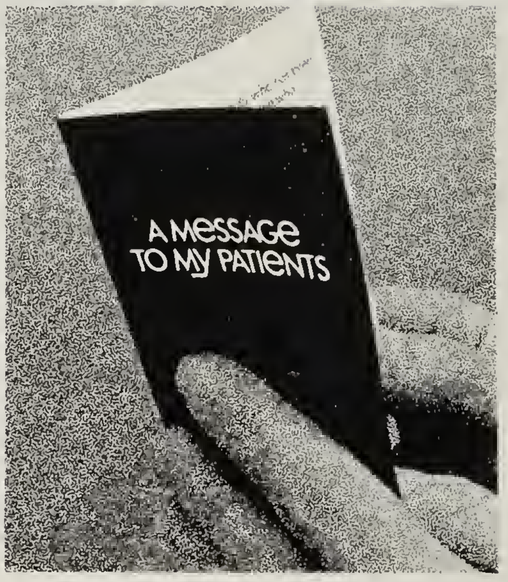
That's why we want your help in reaching people who drink too much too often. As a health-care professional, you can speak with authority and they'll listen. But first they may need some prodding to get them to discuss their drinking habits.



Therefore we're offering you, free of charge, a small (8" x 8") tabletop display card and a supply of pocket-sized leaflets — both titled "A Message to My Patients" — designed to encourage patients to ask your advice about abusive drinking, and to start them thinking about related driving hazards. The materials work. In test cities, up to 60% of physicians displaying them noted increased discussion of alcohol problems

Your local affiliate of the National Council on Alcoholism is also ready to assist you with referrals to counseling and therapy services and in other ways.

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They'll listen to a pro

To: Drunk Driver, Dept. M.D.
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Name______________________________
(Please print or type)

Address______________________________

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Please indicate your medical specialty:____________________

* Or send this coupon to your local affiliate of the National Council on Alcoholism

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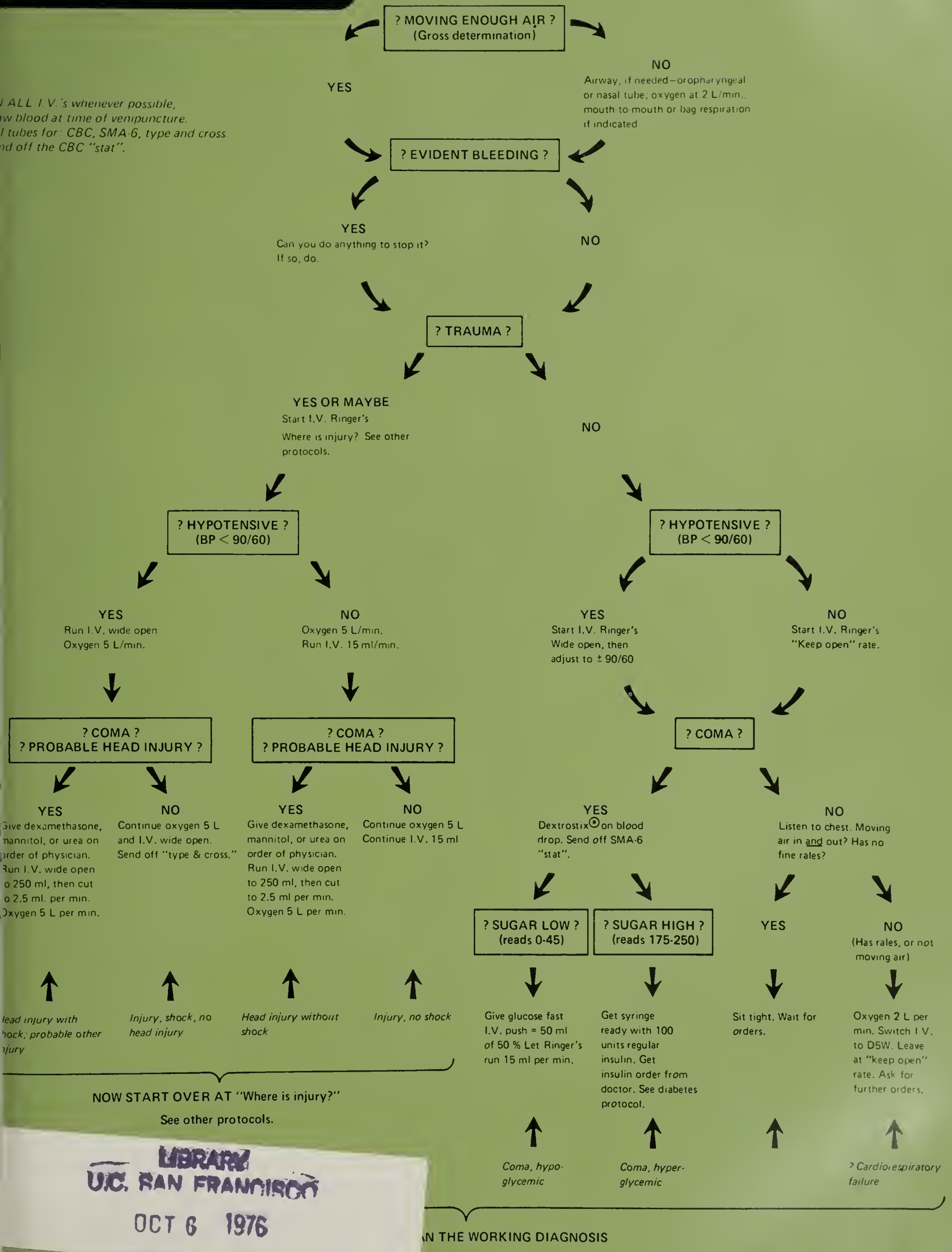

l ALL I.V.'s whenever possible,
w blood at time of venipuncture.
l tubes for: CBC, SMA-6, type and cross.
nd off the CBC "stat".

? MOVING ENOUGH AIR ?
(Gross determination)

YES

NO
Airway, if needed—oropharyngeal or nasal tube; oxygen at 2 L/min., mouth-to-mouth or bag respiration if indicated

? EVIDENT BLEEDING ?

YES
Can you do anything to stop it?
If so, do.

NO

? TRAUMA ?

YES OR MAYBE
Start I.V. Ringer's
Where is injury? See other protocols.

NO

? HYPOTENSIVE ?
(BP < 90/60)

YES
Run I.V. wide open
Oxygen 5 L/min.

NO
Oxygen 5 L/min.
Run I.V. 15 ml/min.

? COMA ?
? PROBABLE HEAD INJURY ?

YES
Give dexamethasone, mannitol, or urea on order of physician.
Run I.V. wide open to 250 ml, then cut to 2.5 ml. per min.
Oxygen 5 L per min.

NO
Continue oxygen 5 L and I.V. wide open.
Send off "type & cross."

? COMA ?
? PROBABLE HEAD INJURY ?

YES
Give dexamethasone, mannitol, or urea on order of physician.
Run I.V. wide open to 250 ml, then cut to 2.5 ml per min.
Oxygen 5 L per min.

NO
Continue oxygen 5 L
Continue I.V. 15 ml

lead injury with hock; probable other njury

Injury, shock, no head injury

Head injury without shock

Injury, no shock

NOW START OVER AT "Where is injury?"
See other protocols.

? HYPOTENSIVE ?
(BP < 90/60)

YES
Start I.V. Ringer's
Wide open, then adjust to ± 90/60

NO
Start I.V. Ringer's
"Keep open" rate.

? COMA ?

YES
Dextrostix® on blood drop. Send off SMA-6 "stat".

? SUGAR LOW ?
(reads 0-45)

Give glucose fast I.V. push = 50 ml of 50 % Let Ringer's run 15 ml per min.

Coma, hypo-glycemic

? SUGAR HIGH ?
(reads 175-250)

Get syringe ready with 100 units regular insulin. Get insulin order from doctor. See diabetes protocol.

Coma, hyper-glycemic

NO
Listen to chest. Moving air in and out? Has no fine rales?

YES
Sit tight. Wait for orders.

NO
(Has rales, or not moving air)
Oxygen 2 L per min. Switch I.V. to D5W. Leave at "keep open" rate. Ask for further orders.

? Cardiorespiratory failure

N THE WORKING DIAGNOSIS

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OCT 6 1976

Both often

Predominant psychoneurotic anxiety

Associated depressive symptoms

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequ
and/ or severity of grand mal seizures
require increased dosage of standard
convulsant medication; abrupt withdr
may be associated with temporary increase in frequency and/ or severity o
seizures. Advise against simultaneous
gestion of alcohol and other CNS depr
sants. Withdrawal symptoms (similar
those with barbiturates and alcohol) h
occurred following abrupt discontinua
(convulsions, tremor, abdominal and
cle cramps, vomiting and sweating). K
addiction-prone individuals under car

respond to one

According to her major
nptoms, she is a psychoneu-
ic patient with severe
kiety. But according to the
cription she gives of her
lings, part of the problem
y sound like depression.
is is because her problem,
hough primarily one of ex-
sive anxiety, is often accom-
nied by depressive symptom-
logy. Valium (diazepam)
n provide relief for both—as
excessive anxiety is re-
ved, the depressive symp-
ns associated with it are also
en relieved.

There are other advan-
es in using Valium for the
nagement of psychoneu-
ic anxiety with secondary
pressive symptoms: the
chotherapeutic effect of
lium is pronounced and
pid. This means that im-
ovement is usually apparent
in the patient within a few days rather than in a week or two, although it may take longer in some patients. In addition, Valium (diazepam) is generally well tolerated; as with most CNS-acting agents, caution patients against hazardous occupations requiring complete mental alertness.

Also, because the psychoneurotic patient's symptoms are often intensified at bedtime, Valium can offer an additional benefit. An *h.s.* dose added to the *b.i.d.* or *t.i.d.* treatment regimen can relieve the excessive anxiety and associated depressive symptoms and thus encourage a more restful night's sleep.

Valium® (diazepam) Ⓘⓥ

2-mg, 5-mg, 10-mg scored tablets

in psychoneurotic
anxiety states
with associated
depressive symptoms

veillance because of their predisposi-
n to habituation and dependence. In
gnancy, lactation or women of child-
ring age, weigh potential benefit
inst possible hazard.
cautions: If combined with other psy-
otropics or anticonvulsants, consider
efully pharmacology of agents em-
yed; drugs such as phenothiazines,
cotics, barbiturates, MAO inhibitors
d other antidepressants may potentiate
action. Usual precautions indicated in
ients severely depressed, or with latent
oression, or with suicidal tendencies.
Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Possibly Effective:

1. For the relief of symptoms associated with cerebral vascular insufficiency.
2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.
3. Threatened abortion.

Final classification of the less-than-effective indications requires further investigation.

Composition: Vasodilan tablets, isoxsuprine HCl, 10 mg. and 20 mg.

Dosage and Administration: 10 to 20 mg. three or four times daily.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.

Adverse Reactions: On rare occasions, oral administration of the drug has been associated in time with the occurrence of severe rash. When rash appears, the drug should be discontinued. Occasional overdosage effects such as transient palpitation or dizziness are usually controlled by reducing the dose.

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* **Warning**
This fixed combination drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual patient. If the fixed combination represents the dosage so determined, its use may be more convenient in patient management. The treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** *Edema:* That associated with congestive heart failure, cirrhosis of the liver, the nephrotic syndrome; steroid-induced and idiopathic edema; edema resistant to other diuretic therapy. *Mild to moderate hypertension:* Usefulness of the triamterene component is limited to its potassium-sparing effect.

Contraindications: Pre-existing elevated serum potassium. Hypersensitivity to either component. Continued use in progressive renal or hepatic dysfunction or developing hyperkalemia.

Warnings: **Do not use dietary potassium supplements or potassium salts unless hypokalemia develops or dietary potassium intake is markedly impaired.** Enteric-coated potassium salts may cause small bowel stenosis with or without ulceration. Hyperkalemia (>5.4 mEq/L) has been reported in 4% of patients under 60 years, in 12% of patients over 60 years, and in less than 8% of patients overall. Rarely, cases have been associated with cardiac irregularities. Accordingly, **check serum potassium during therapy, particularly in patients with suspected or confirmed renal insufficiency (e.g., elderly or diabetics). If hyperkalemia develops, substitute a thiazide alone. If spironolactone is used concomitantly with 'Dyazide', check serum potassium frequently**—both can cause potassium retention and sometimes hyperkalemia. Two deaths have been reported in patients on such combined therapy (in one, recommended dosage was exceeded; in the other, serum electrolytes were not properly monitored). Observe patients on 'Dyazide' regularly for possible blood dyscrasias, liver damage or other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving Dyrenium (triamterene, SK&F). Rarely, leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with the thiazides. Watch for signs of impending coma in acutely ill cirrhotics. Thiazides are reported to cross the placental barrier and appear in breast milk. This may result in fetal or neonatal hyperbilirubinemia, thrombocytopenia, altered carbohydrate metabolism and possibly other adverse reactions that have occurred in the adult. When used during pregnancy or in women who might bear children, weigh potential benefits against possible hazards to fetus.

Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in postsympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis; rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting (may indicate electrolyte imbalance), diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 capsules; in Single Unit Packages of 100 (intended for institutional use only).

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

*INDICATIONS. Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: Management of nausea and vomiting and dizziness associated with motion sickness.

Possibly Effective: Management of vertigo associated with diseases affecting the vestibular system.

Final classification of the less than effective indications requires further investigation.

CONTRAINDICATIONS. Administration of Antivert (meclizine HCl) during pregnancy or to women who may become pregnant is contraindicated in view of the teratogenic effect of the drug in rats.

The administration of meclizine to pregnant rats during the 12-15 day of gestation has produced cleft palate in the offspring. Limited studies using doses of over 100 mg./kg./day in rabbits and 10 mg./kg./day in pigs and monkeys did not show cleft palate. Congeners of meclizine have caused cleft palate in species other than the rat.

Meclizine HCl is contraindicated in individuals who have shown a previous hypersensitivity to it.

WARNINGS. Since drowsiness may, on occasion, occur with use of this drug, patients should be warned of this possibility and cautioned against driving a car or operating dangerous machinery.

Usage in Children: Clinical studies establishing safety and effectiveness in children have not been done; therefore, usage is not recommended in the pediatric age group.

Usage in Pregnancy: See "Contraindications."

ADVERSE REACTIONS. Drowsiness, dry mouth and, on rare occasions, blurred vision have been reported.

More detailed professional information available on request.

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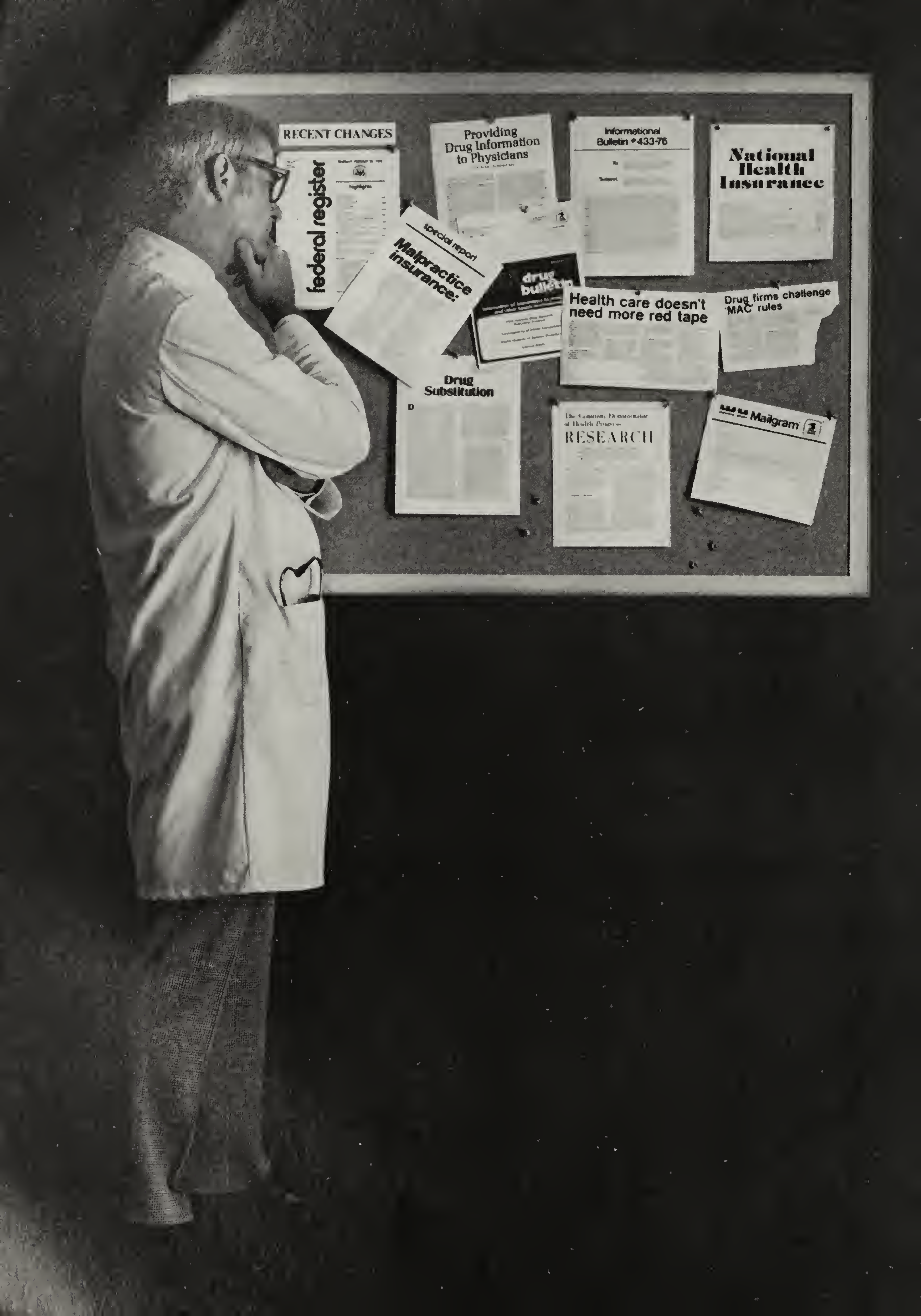
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THERE ARE A LOT OF PEOPLE GETTING BETWEEN YOU AND YOUR PATIENT.

Medicine today is in the spotlight, subjected to all kinds of scrutiny. Your control over patient therapy is being monitored, judged and occasionally abrogated, sometimes by unknown third parties.

The worry is that in the wake of this focus, the relationship between you and your patient will be weakened, without offsetting benefits. Consider three examples:

Drug substitution In most states, pharmacy laws, regulations or professional custom stipulate that your non-generic prescriptions be filled with the precise products you prescribe. But in the last five years, a dozen or more State laws have been changed, permitting the pharmacist in most cases to select a product of the same generic drug to fill any prescription.

Ironically, this dilution of physician control has taken place against a background of growing evidence that purportedly equivalent drug products may be inequivalent, since neither present drug standards nor their enforcement are optimal. In fact, the FDA itself says it has not enforced the same standards for hundreds of "follow-on" products that it had applied to the original NDA approvals. Thus physician control over patient therapy is being eroded with a risk that patients may be exposed to drugs of uncertain quality.

The major advertised claim for substitution is reduced prescription prices for consumers. Yet no documentation of any significant savings has been produced.

MAC Maximum Allowable Cost, MAC for short, is a Federal regulation designed to cut the Government's drug bill by setting price ceilings for drugs dispensed to Medicare and Medicaid patients. Unless the prescriber certifies on the prescription that a particular product is medically necessary, the Government intends to pay only for the cost of the lowest-priced, purportedly-equivalent, generally-available product. The effect of the program may be that elderly and indigent patients will be restricted to products which someone in Washington believes are priced right. Practicing doctors will have little to say about administration of the program, since Government will have absolute authority to make its choices stick.

The drug lag The future of drug and device research depends upon a scientific and regulatory environment that encourages therapeutic innovations. The American pharmaceutical industry annually is spending more than $1 billion of its own funds and evaluating more than 1,200 investigational compounds in clinical research. Disease targets include cancer, atherosclerosis, viruses and central nervous system disorders, among others. But there is a major barrier to the flow of new drugs to your patients: The cost of the research is more than ten times what it was, per product, in 1962; and whereas governmental clearance of new drug applications took six months then, it commonly consumes two years now.

The FDA needs adequate time, of course, to consider data. But it is equally clear that the present approval process contributes to needless delay of needed therapy. That's why the increased efficiency of the drug approval process is vital to all our futures.

If these issues concern you, we suggest that you make your voice heard—among your colleagues and your representatives in State legislatures and in Washington.

It could make a difference in your practice tomorrow.

P·M·A

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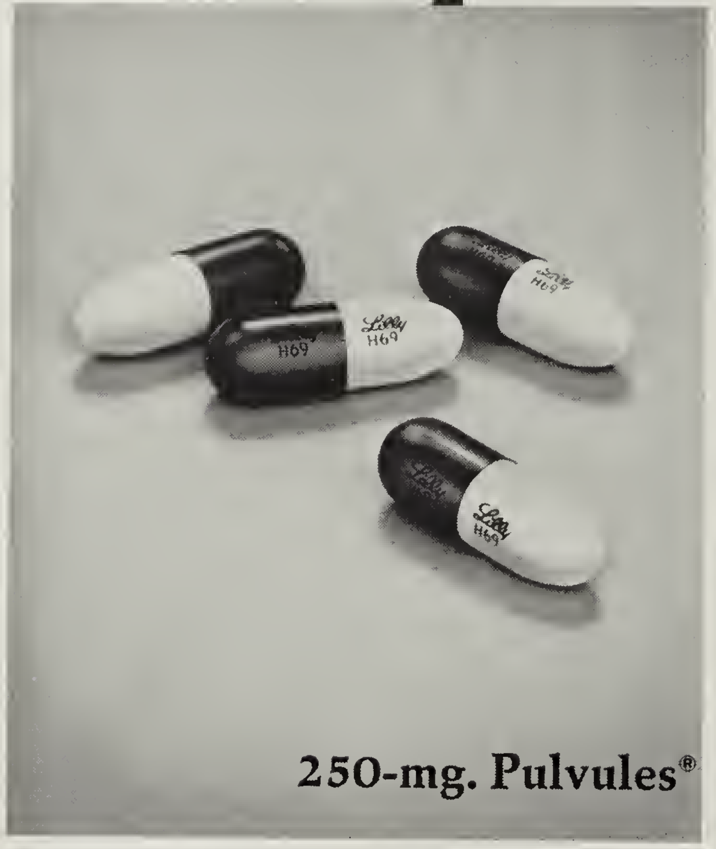

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Original Articles

An Ingenious, Easily Read Presentation Of The Diagnostic Maneuvers, Pro And Con, Laparotomy After Abdominal Trauma. A very Extensive Bibiliography And Several Easy To Follow Charts are Also Offered.

ABDOMINAL TRAUMA: WHEN TO GO IN WHEN TO STAY OUT?

DOUGLAS LINDSEY, M. D., DR. P. H.
MARTIN SILVERSTEIN, M. D.

Definitive diagnosis of the type, location, and extent of intra-abdominal injury is usually established only at laparotomy. Decision on when to go in, or whether to stay out, is less precise, and most often depends on the running sum of a variety of considerations. Sound guidelines can be developed for minimizing both the delay before needed intervention, and the hasty but unnecessary laparotomy.

Less than two out of three stab wounds of the abdomen produce visceral damage which demands operative intervention.[1-3] Picking out the third case—the one which does not need laparotomy—is a neat trick, if you can do it consistently. Not so dramatically neat, but far more important, is sifting through the Emergency Room flood of more diffuse trauma—collisions, falls, beatings—to pick up the closed abdominal injury. Medical economics calls for a reasonable minimum of unnecessary laparotomies; medical excellence permits us no false negative diagnoses, and demands decision with minimum delay.

The immediate general care of the accident victim in the Emergency Room is a relatively straightforward proposition (Fig. 1). If there is any reasonable possibility of intra-abdominal injury then further concerted study is required (Figs. 2,3). "Reasonable possibility" includes:

1. Visible injury: contusion, laceration, abrasion, or puncture — anywhere between the nipples and the gluteal folds; fractures of pelvis or lower ribs.
2. Mechanism of injury specific enough to incite suspicion.
3. Severity of injury sufficient to cause multiple system damage.
4. Any injured patient who is too far out to give a good history.
5. Persistent shock, not otherwise explainable, responding at least transiently to saline solutions.

Penetrating thoracic injury, or major closed thoracic trauma should raise immediate suspicion of concomitant abdominal lesions.[4,5] Any visible mark of a seat belt indicates major force applied to the abdomen.[6-9]

Detailed knowledge of direction and magnitude of applied force is of predictive value,[10,11] but it is normally sufficient to deal with generalities,[12] such as the fact that a driver who bangs his head on the windshield, or his knee on the dash, almost certainly impacted the steering wheel with his chest or abdomen. Occasionally the loose association of specific organ damage with a particular type of injury is a pointed clue;

Submitted for publication 5/76.

From: Dept. of Surgery, U of A, Tucson, AZ 85724. (Dr. Lindsey and Dr. Silverstein). Reprint requests to Dr. Lindsey. The stimulus for development of the algorithm presented herein originated in a seminar presented at Navapache Hospital, Lakeside, under sponsorship of the Continuing Education Service Area Program. Manuscript abridged from presentation on same subject at Arizona Medical Association Annual Meeting, 24 April, 1975.

ARIZONA ALGORITHMS IN EMERGENCY MEDICINE

Initial Management of the Major Emergency
(Standing orders, first 20 minutes,
paramedics and ER staff)

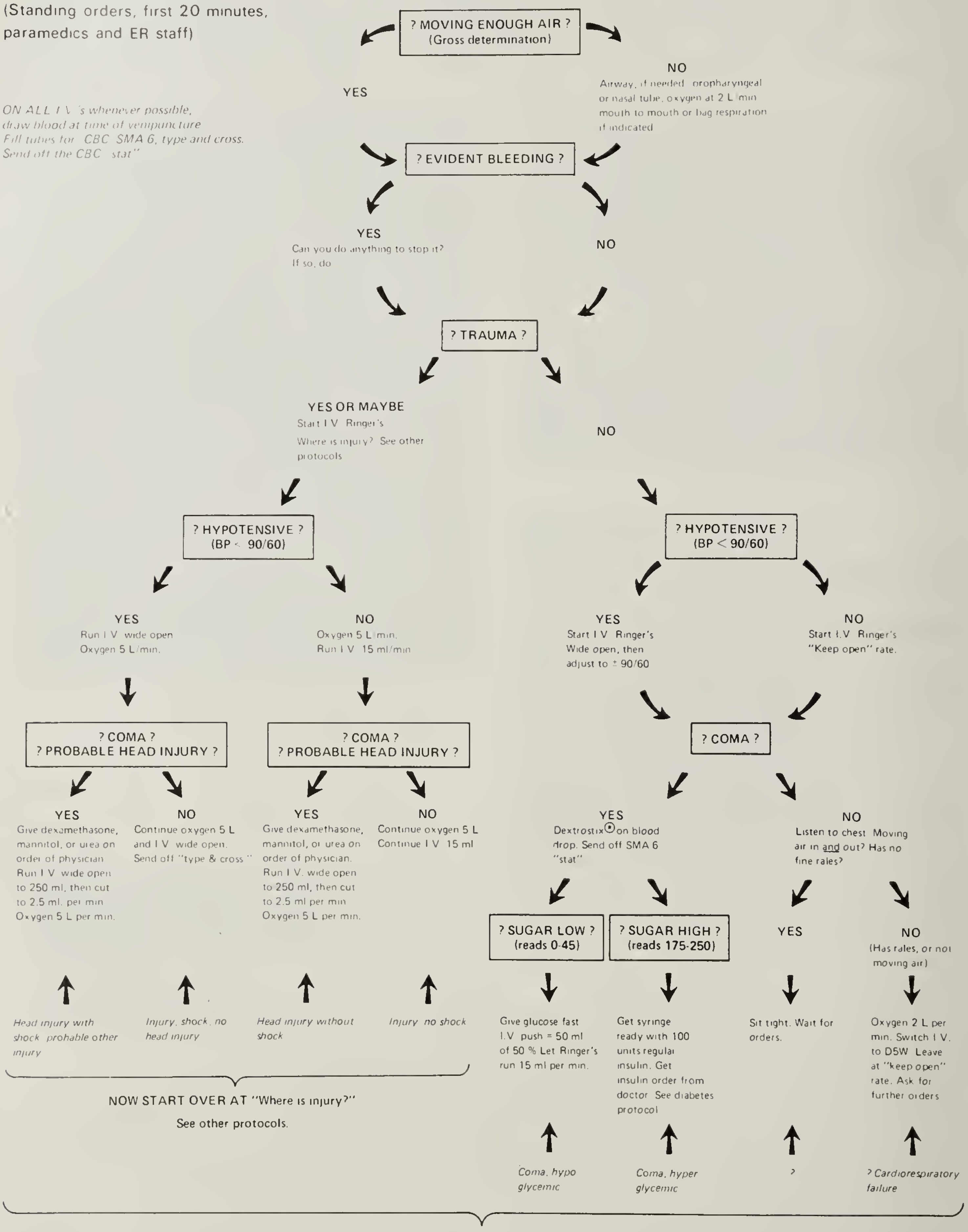
ON ALL I V 's whenever possible,
draw blood at time of venipuncture
Fill tubes for CBC SMA 6, type and cross.
Send off the CBC "stat"
? MOVING ENOUGH AIR ?
(Gross determination)
YES
NO
Airway, if needed oropharyngeal or nasal tube, oxygen at 2 L/min mouth to mouth or bag respiration if indicated
? EVIDENT BLEEDING ?
YES
Can you do anything to stop it? If so, do
NO
? TRAUMA ?
YES OR MAYBE
Start I V Ringer's
Where is injury? See other protocols
NO
? HYPOTENSIVE ?
(BP < 90/60)
YES
Run I V wide open
Oxygen 5 L/min.
NO
Oxygen 5 L/min.
Run I V 15 ml/min
? COMA ?
? PROBABLE HEAD INJURY ?
YES
Give dexamethasone, mannitol, or urea on order of physician Run I V wide open to 250 ml, then cut to 2.5 ml. per min Oxygen 5 L per min.
NO
Continue oxygen 5 L and I V wide open. Send off "type & cross"
Head injury with shock probable other injury
Injury, shock, no head injury
? COMA ?
? PROBABLE HEAD INJURY ?
YES
Give dexamethasone, mannitol, or urea on order of physician. Run I V. wide open to 250 ml, then cut to 2.5 ml per min Oxygen 5 L per min.
NO
Continue oxygen 5 L
Continue I V 15 ml
Head injury without shock
Injury no shock
NOW START OVER AT "Where is injury?"
See other protocols.
? HYPOTENSIVE ?
(BP < 90/60)
YES
Start I V Ringer's Wide open, then adjust to ± 90/60
NO
Start I.V Ringer's "Keep open" rate.
? COMA ?
YES
Dextrostix⊙on blood drop. Send off SMA 6 "stat"
? SUGAR LOW ?
(reads 0-45)
Give glucose fast I.V push = 50 ml of 50 % Let Ringer's run 15 ml per min.
Coma, hypo glycemic
? SUGAR HIGH ?
(reads 175-250)
Get syringe ready with 100 units regular insulin. Get insulin order from doctor See diabetes protocol
Coma, hyper glycemic
NO
Listen to chest Moving air in and out? Has no fine rales?
YES
Sit tight. Wait for orders.
?
NO
(Has rales, or not moving air)
Oxygen 2 L per min. Switch I V. to D5W Leave at "keep open" rate. Ask for further orders
? Cardiorespiratory failure
TELL PHYSICIAN THE WORKING DIAGNOSIS

Figure 1

the loose association of specific organ damage with a particular type of injury is a pointed clue; for example, the handlebar of a bike is a notorious cause of pancreatic trauma in children*

Extra-abdominal lesions which may accompany abdominal trauma are about evenly divided between the extremities, the chest, and the central nervous system. Besides the general effect of multiple injury in increasing mortality any one of the other injuries may be more obvious (even if less serious) than the abdominal injury, thus unfairly pre-empting initial attention.[13-15]

"Ordinary physical examination is extremely precise in the evaluation of the patient with blunt abdominal injury who is conscious and responsive."[16] On this point there is some dissent, and we stand with those who are less than completely confident. There is near unanimity of opinion on the converse: put little faith in the clinical findings if the patient is *not* responsive and cooperative.

One of the most serious errors you can make in the initial management of multiple trauma is to neglect the abdomen by attributing persistent shock to a head injury. Head injury does not cause profound hypotension until the patient is nearly terminal and functioning at a medullary level, a state which can be readily confirmed by simple physical examination. If there is no clear accounting for the hypotension through external blood loss; if there is not a massive hemothorax, a cardiac tamponade, or the unmistakable shadow of a ruptured aorta; if the femur is fractured unilaterally and not bilaterally — then you must assume that the patient is bleeding into or behind the peritoneal cavity (pelvic; retroperitoneal), particularly if there is a significant pressure response to the rapid infusion of a liter or two of a saline solution.[17-20]

If, on any of the above grounds, there is suspicion of abdominal injury, then add two simple items to your immediate data base: serum amylase, and a girth measurement. Neither is of immediate help. Looking at the abdomen two hours from now and thinking that it is getting fuller is no help either, but a measured 2 cm increase in the waistline at the umbilicus can be. Serum amylase is rarely elevated immediately after injury, even after direct injury to the pancreas; a later rise may be significant.[21-23]

If rupture of an intra-peritoneal organ is evident on signs and symptoms no further diagnostic measures are demanded. Abdominal x-rays can add or subtract little to a decision already made for surgical intervention. If the decision is not yet made then we need all the help the radiologist can give us. As a general rule we do not order portable abdominal films in cases of suspected trauma. Portable chest films, yet; but the radiographic signs of abdominal trauma are subtle, and films of top quality are essential: supine and erect if possible, left lateral recumbent or cross table lateral if not.[24] If the patient is in good enough shape to go to radiology with an attendant and with I. V.'s running, then send him. If his condition is too poor for that then maybe he needs to go to the operating room instead.

Detection of small amounts of free air by pooling it under the diaphragm is the prime reason for the horizontal beam films. Air can, of course, enter the abdomen through a penetrating injury of the abdominal wall in the absence of visceral injury, and it occasionally enters via the fallopian tubes, but for all practical purposes the radiographic demonstration of free retroperitoneal gas bubbles, is tantamount to the diagnosis of a perforated hollow viscus. The opposite does not hold; there may be a major perforation of the gastrointestinal tract with no free air demonstrable.[25,26]

The radiographic signs of blood in the peritoneal cavity or retroperitoneal space are far less clear than is demonstration of free air or gas bubbles. Two passing reminders: put little faith in the significance of psoas shadows, present or absent;[27,28] and remember that the spleen and the pancreas share a common blood supply—evidence of injury of one should raise suspicion of injury to the other.[29-32]

Continued bleeding is a threat to life of greater magnitude than is peritonitis.[33,34] A "negative" abdominal series is no amelioration of that threat. If you are losing ground, open the belly while you still have time. If there are signs suggestive of beginning intra-peritoneal inflammation, admit for close and continuous watch. If a thorough review turns up no evidence of either beginning irritation or continuing bleeding, then send the patient home after his other injuries are cared for.

An early decision one way or another may not be possible; it takes several hours for the hematocrit to fall, and the white blood count to rise.[35,36] It may take longer than that before the tenderness of a peritoneal irritative process localizes, spreads, or changes position to make it

ARIZONA ALGORITHMS IN EMERGENCY MEDICINE

Management of Abdominal Trauma

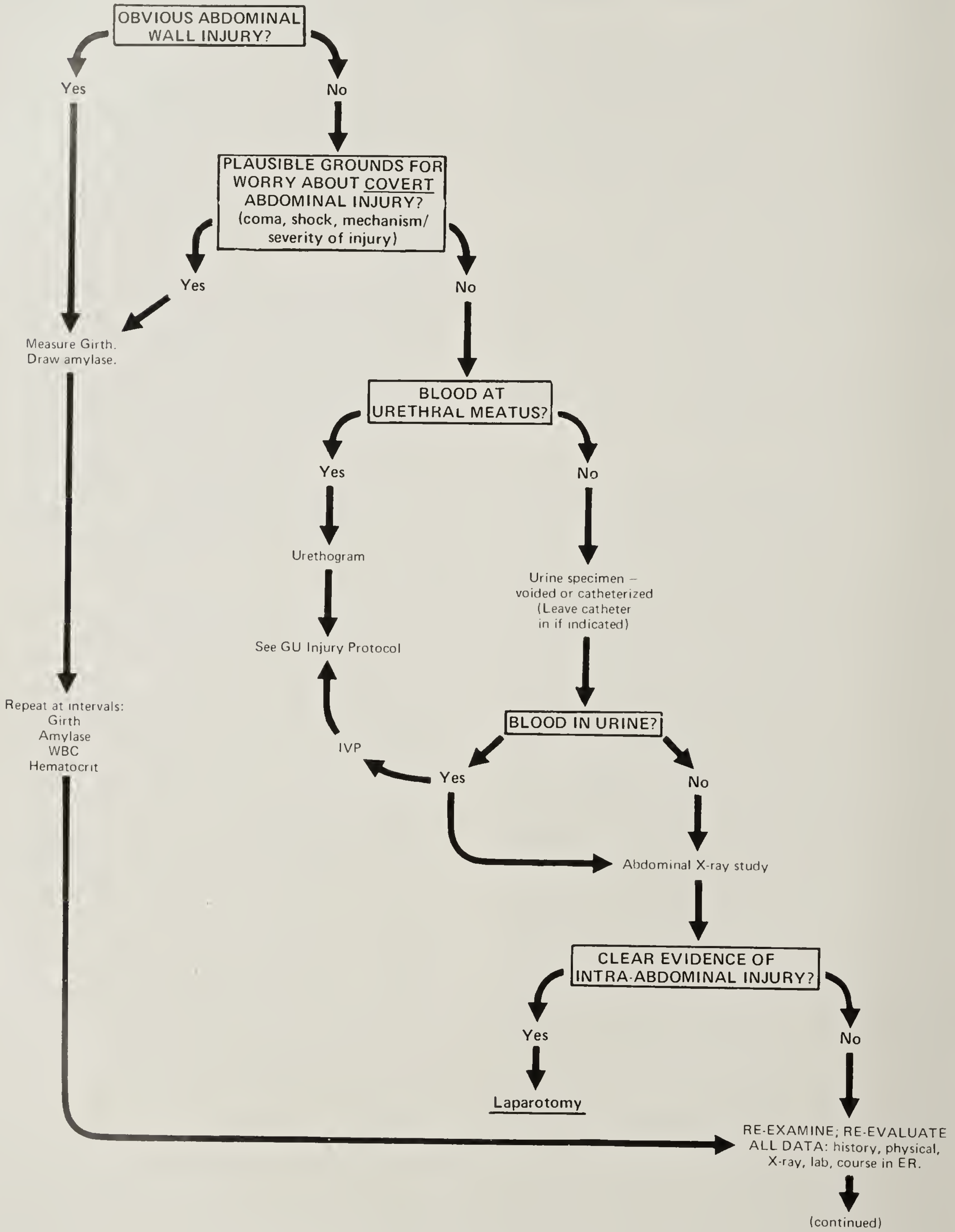
OBVIOUS ABDOMINAL WALL INJURY?
Yes
No
PLAUSIBLE GROUNDS FOR WORRY ABOUT COVERT ABDOMINAL INJURY? (coma, shock, mechanism/ severity of injury)
Yes
No
Measure Girth. Draw amylase.
BLOOD AT URETHRAL MEATUS?
Yes
No
Urethogram
See GU Injury Protocol
Urine specimen – voided or catheterized (Leave catheter in if indicated)
Repeat at intervals: Girth Amylase WBC Hematocrit
BLOOD IN URINE?
IVP
Yes
No
Abdominal X-ray study
CLEAR EVIDENCE OF INTRA-ABDOMINAL INJURY?
Yes
No
Laparotomy
RE-EXAMINE; RE-EVALUATE ALL DATA: history, physical, X-ray, lab, course in ER.
(continued)

Figure 2

ARIZONA ALGORITHMS IN EMERGENCY MEDICINE
Management of Abdominal Trauma, continued

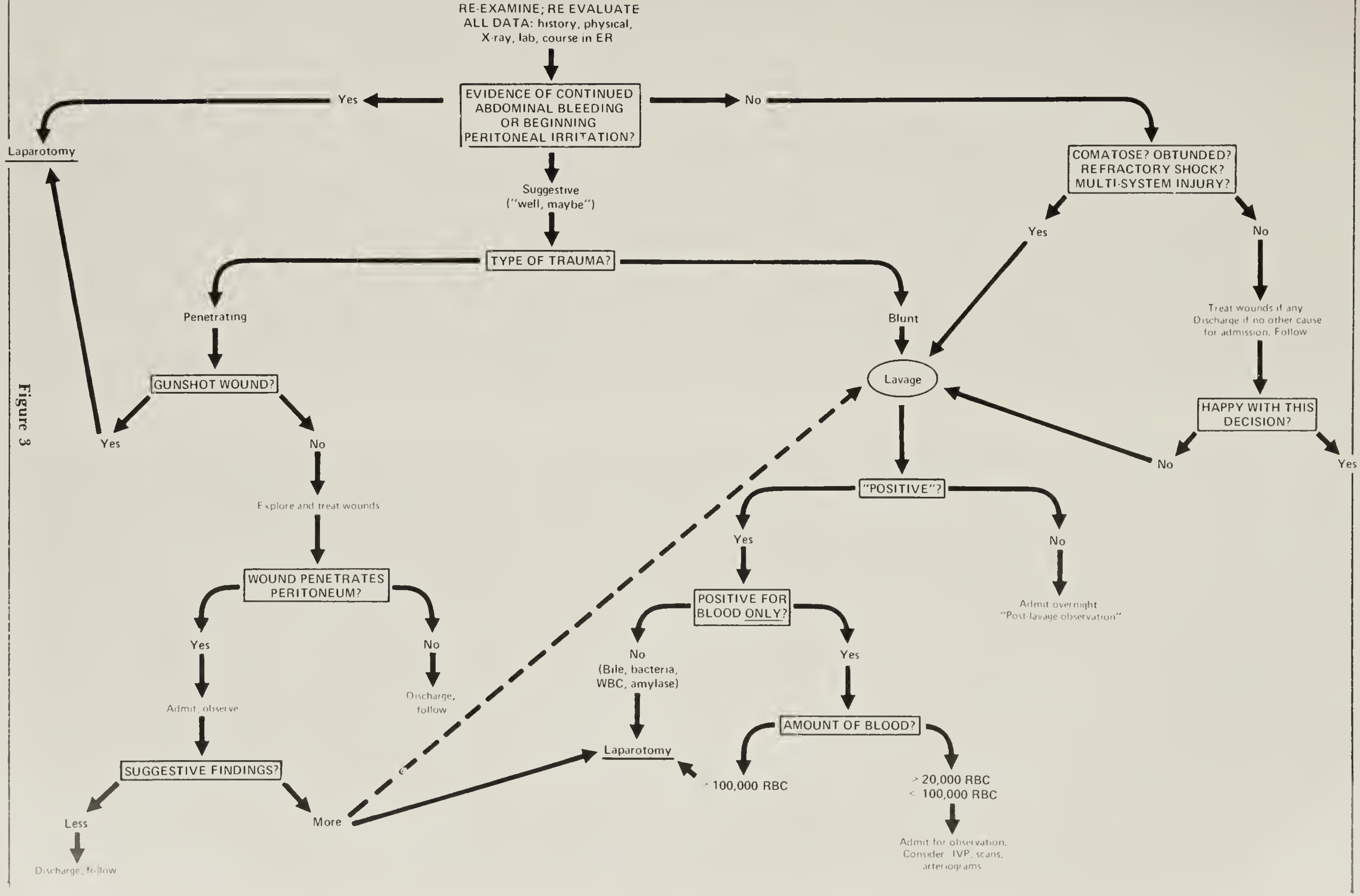
RE-EXAMINE; RE EVALUATE ALL DATA: history, physical, X-ray, lab, course in ER
EVIDENCE OF CONTINUED ABDOMINAL BLEEDING OR BEGINNING PERITONEAL IRRITATION?
Yes
Laparotomy
No
COMATOSE? OBTUNDED? REFRACTORY SHOCK? MULTI-SYSTEM INJURY?
Yes
No
Treat wounds if any Discharge if no other cause for admission. Follow
HAPPY WITH THIS DECISION?
No
Yes
Suggestive ("well, maybe")
TYPE OF TRAUMA?
Penetrating
Blunt
Lavage
GUNSHOT WOUND?
Yes
No
Explore and treat wounds
WOUND PENETRATES PERITONEUM?
Yes
Admit, observe
No
Discharge, follow
SUGGESTIVE FINDINGS?
Less
Discharge, follow
More
"POSITIVE"?
Yes
No
Admit overnight "Post-lavage observation"
POSITIVE FOR BLOOD ONLY?
No (Bile, bacteria, WBC, amylase)
Laparotomy
Yes
AMOUNT OF BLOOD?
> 100,000 RBC
> 20,000 RBC < 100,000 RBC
Admit for observation. Consider IVP, scans, arteriograms

Figure 3

stand out from the background of abdominal contusion;[37-38] a perforated stomach speaks out promptly, blood is a weak irritant, clean urine may cause no problem for days.

When you feel that you cannot make a confident decision to go in or stay out there are two things you can do: admit and observe; or tap the belly. If observation is elected then continuity of observer is important; transfer responsibility only at a joint examination on change of the watch. When doubt remains, tap and lavage.[39-44]

The past decade has seen a steady sequence of changes in the procedure customarily followed for performing diagnostic peritoneocentesis: from a single tap, to all four quadrants; from taps with a cannula to placement of a dialysis catheter; from insertion by trocar to insertion through a low midline incision. We are now only a step away from minilaparotomy. Why not open the abdomen a few centimeters and have a good look?[45-46]

There is little doubt that placement of the larger catheter, with multiple drainage holes, and instillation of a significant volume of fluid will lessen the chances of a false negative tap. There is less certainty about the improvement of the error rate in terms of false positives.[47-49] Incidentally, data on the rate of false negatives—the true index of reliability—are usually understated on the basis of the number of false negatives in the total series; they should preferably be calculated as percentage of false negatives among the number of taps read as negative only.[50-55]

Something approaching a consensus on when a tap is "positive" is reached at 100,000 mm^3 in aliquot of returned fluid. This reflects a nominal 20 ml of blood washed out in the liter of instilled saline or glucose, enough to impart a bright red color when returned to the flask, but only a light rose in the thinner column seen in the tubing. As this rose deepens almost imperceptibly with increasing amounts of blood there is a remarkably abrupt cut-off point (at about 100,000 RBC/mm^3) when newsprint seen through the tubing becomes illegible. When you cannot read the print the tap is "strongly positive"; if you can still read it, but the fluid is pink, the tap is "weakly positive"; if there is no color at all you can tentatively label it "negative", but two caveats are in order. First, don't pay all of your attention to red cells; a tap is abnormal ("positive") if the returned fluid tests high for amylase, counts out more than 1000 white cells per mm^3, is positive for bile, or shows any bacteria in the spun sediment.[56] Second, the cutoff at 100,000 is *statistical*; if you open all injured abdomens which return counts at or above that figure you will find that 90% or more of them needed something done with the abdomen. On the other hand, as many as a third of patients with taps below 100,000 RBC/mm^3 will subsequently develop findings warranting laparotomy.[39, 44]

We have two suggestions on avoiding the pitfall of the false negative. First, never do a lavage until you have already made a clear decision on what you would do if a tap could not be performed. If your decision would be to operate, then don't let a negative or weakly positive tap talk you out of it. If your decision would be to observe and the tap is negative, then continue to observe, with care. Let a positive tap change your mind, but not a negative one. Second, adopt the policy of overnight admission, "post-lavage observation."

You will always be faced with the patient who fits neither of the definitive groups: "discharge", or "operate". Further studies are required. Arteriography can elucidate many abdominal injuries which are otherwise manifested only by bleeding which of itself is no threat: ceased, minor, moderate, or entrapped in some tissue. But arteriography is an expensive and invasive procedure. We feel that in time it will give way in great part to more extensive use of echosonography, and of radioisotopic scans.[57-61] Wider experience with scans and sonos will probably provide us with a sound basis for confident, selective non-intervention in closed trauma to the liver and spleen. We believe with others[62, 63] that the spleen should not be treated as an expendable organ, and we are willing to temporize with minor lacerations even though we are not ready yet to espouse suturing the spleen[64, 65] or gluing it back together.[66] We feel that suture of minor lacerations of the liver which have ceased to bleed is meddlesome surgery.

You may disagree. These are personal judgments, and personal preference and experience weighs heavily in how to treat specific injuries once the abdomen is opened. Indeed, the same personal judgment applies to the decision for or against laparotomy. Our algorithm—in spite of its bold lines and precise labeling—is a framework to hang your own thoughts on, not a box to hem them in. We hope that it is a help in expediting and facilitating a confident decision: Stay out? or Go in?

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THE USE OF HOMOGENEOUS BONE TRANSPLANTS IN THE TREATMENT OF LOCALLY MALIGNANT BONE TUMORS: A CASE REPORT

ROBERT G. VOLZ, M. D., F.A.C.S.
JOSEPH C. NICHOLS, M. D.

The treatment of large locally destructive bone lesions which have produced an extensive loss of cortical bone, frequently poses a difficult therapeutic problem for the orthopedic surgeon.

In many cases when evidence of metastatic spread is lacking, the locally aggressive nature of the lesion demands en bloc resection. The use of an autogenous bone graft to stabilize the resected defect in such situations is frequently inadequate. The surgeon is then left with the following options: 1) primary amputation, 2) en bloc resection followed by prosthetic replacement, 3) en bloc resection with homograft transplant. The treatment of large locally destructive lesions which have violated the cortical shell by irradiation alone is unacceptable as the patient is left with a skeletal defect which will give rise to instability, deformity, pain, and an overall functional disability. Recently at the Arizona Medical Center, a large locally destructive lesion of the distal femur was treated by resection of the distal half of the femur and replacement with a tibial homograft (Figure 1).

CASE REPORT

D. E., a 32 year old Mexican national, mother of six children, was initially seen at the Arizona Medical Center in March, 1975, with complaints of eight months duration of pain and swelling confined to the distal left femur. In July of 1974 the patient had sought medical care elsewhere for pain confined to her left knee following a fall. X-ray examination in July, 1974, revealed a large, eccentrically located, lytic lesion of the distal femur consistent with a giant cell tumor of bone. The patient had declined biopsy or other care at that time, but because of increasing disability the patient had returned to her primary care physician some seven months later in February, 1975. X-rays at this time revealed further enlargement to the area of bone destruction with significant loss of cortical bone distally. Tissue obtained by an open biopsy was consistent with the diagnosis of giant cell tumor, grade II. The patient was referred to the Arizona Medical Center for further care.

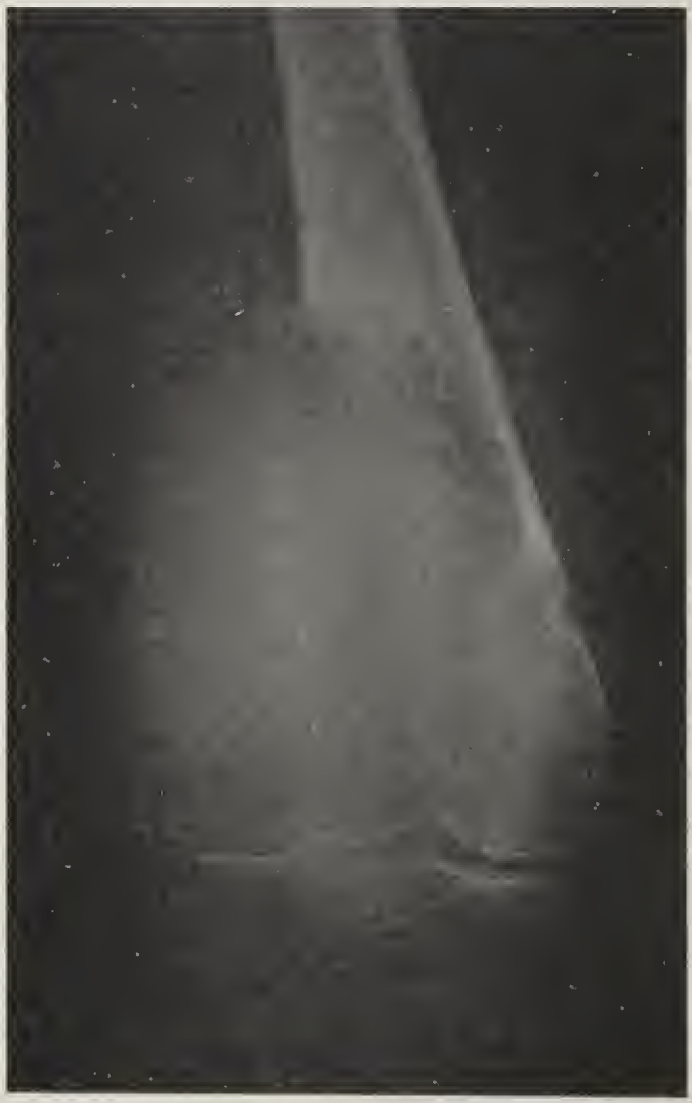

Figure 1

Radiographic appearance of the destructive lesion involving the distal left femur. The medial cortex has been violated by the neoplastic process.

Submitted for publication 3 76.

From: Department of Surgery, Section of Orthopedic Surgery, Arizona Medical Center, Tucson, Arizona, 85724. Reprint requests to Dr. Volz.

Physical examination in March, 1975, disclosed an enlarged, warm, tender, swollen left knee. Motion was guarded and limited. The patient required the use of two crutches to ambulate. Bone scanning and chest laminograms revealed no evidence of metastatic involvement. Surgical amputation of the extremity at the level of the midthigh was recommended. This was adamantly refused by the patient. Because of the extension of the tumor into the quadriceps mechanism, en bloc resection with prosthetic replacement seemed ill-advised. En bloc resection with homograft transplant was decided upon.

Surgically, the lesion was approached by a large U-shaped incision which extended distally to the level of the tibial tuberosity. The patellar tendon was transsected and extension of the tumor into the suprapatellar pouch noted. An anterior flap, superficial to the quadriceps, was developed to the midthigh level. At this point the femur was transsected and the en bloc resection continued posteriorly to include the posterior musculature, but sparing the neurovascular bundle. The specimen was then amputated through the tibial plateau just proximal to the tibial tubercle.

A homograft consisting of a distal tibia had been harvested approximately one week preoperatively from a patient who had died of cardiovascular disease. The collection of the homograft was performed under sterile conditions; the homograft had been placed in an antibiotic solution of Bacitracin and Neomycin and had been frozen at -20°C. At the time of implantation as much of the soft tissue and marrow was removed as was possible.

Immediately prior to transplantation, the tibial plateau of the homograft was amputated by use of a reciprocating saber saw. The homograft was then inserted in an inverted fashion, replacing the amputated distal femoral segment. A Kuntschner nail, which had been pre-bent 7° to provide for a slight amount of flexion at the knee, was then inserted in a retrograde fashion through the femoral canal and was driven proximally through a separate gluteal incision. The nail was then driven distalward through the medullary canal to include the graft until it was seated distally within the proximal portion of the patient's tibia (Figure 2). Following routine wound closure, the extremity was placed in a bulky dressing with splints.

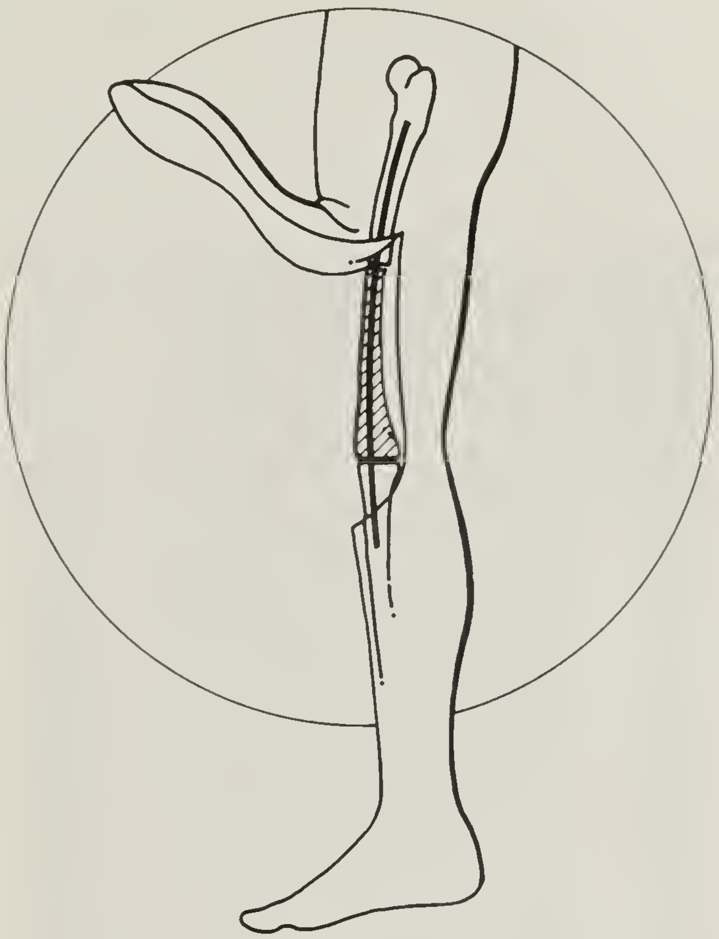

Figure 2

Diagram of the inverted tibial homograft following its insertion into the area created by resection of the tumor mass.

Microscopic examination of the lesion revealed extension of the tumor into cartilaginous regions of the medial tibial plateau, tendons of the semimembranousus, gracilis, and joint capsule in addition to the quadriceps mechanism. Margins of the resected lesion were free of tumor cells.

Postoperatively, the patient developed an area of skin loss in the region of the previous biopsy incision, which had of necessity been included in the operative skin flap. Subsequently a bipedicle skin flap was used to cover the defect with eventual good wound healing. The patient was started on partial weight bearing with crutches five months postoperatively. When last seen in follow-up at 12 months postoperatively, the patient had no knee pain, and was ambulating with one crutch. X-rays at that time disclosed callus formation at both the proximal and distal levels of the homograft anastomosis. The homograft remained structurally intact (Figure 3).

Discussion: The treatment of large locally destructive bone lesions by the use of homogeneous bone transplants was first undertaken by McEwen in 1870[7]. Since that time numerous other physicians have studied the effects of transplanting large bone homografts. Axhausen

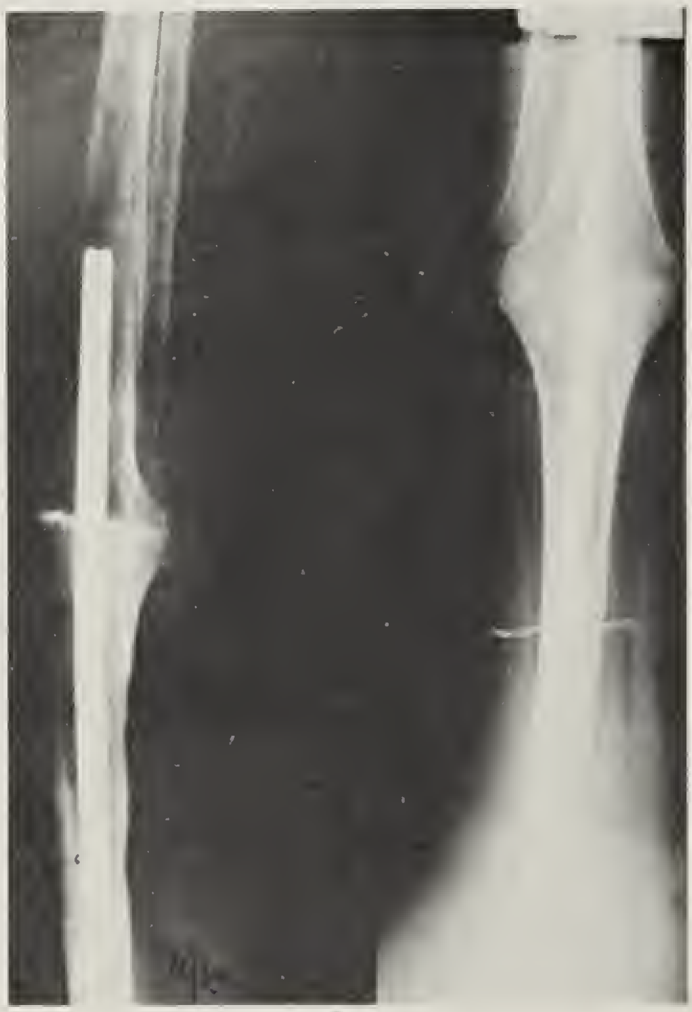

Figure 3

Radiographic appearance of the inverted tibial homograph. Fixation was achieved by use of a 48 cm Kuntcher intramedullary nail.

(1908) described the biological fate of bone grafts and introduced the concept of creeping substitution.[1] This concept of resorption of bone grafts with appositional new bone formation has stood the test of time with little modification to the present day. In 1942 Inclan introduced the concept of the bone bank[4]. The use of bone grafts has since become a well-accepted procedure for the orthopedic surgeon; the use of massive homograft bone transplants has remained limited, and their indications poorly understood.

Biologically, the response of both host and homograft following implantation has been extensively studied.[2 3 4 5 8 12 16 17 26] Histologically, the osteocytes in homografts, just as with autogenous grafts, quickly die following transplantation; complete marrow necrosis of the graft is also noted within 24 hours.[12] Thus the massive homograft must be considered as a scaffold devoid of its own living cellular elements until new bone arising from the host appears. This cellular invasion of the graft, creeping substitution, is dependent upon the size or surface area of the graft and may require years before it is complete. Transplantation of cartilaginous elements results in death to the chondrocytes with a loss of staining characteristics to the hyaline cartilage.[5 6] A fibrous matrix generally replaces the articular surface.

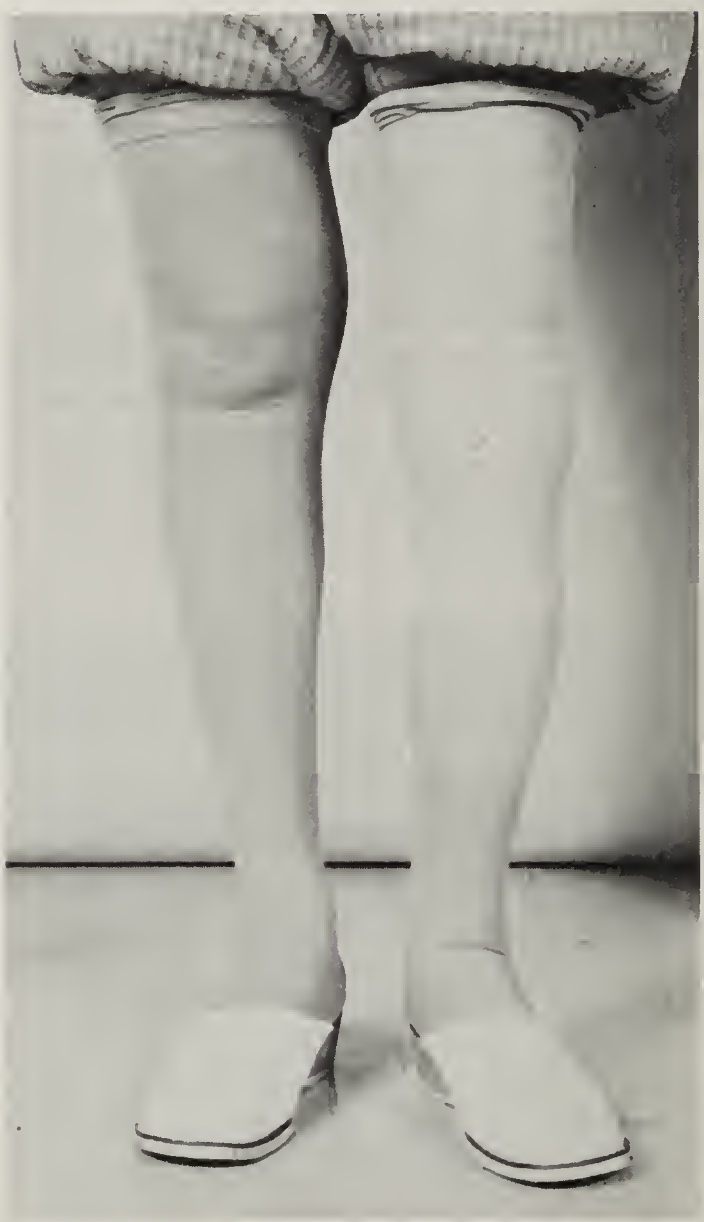

Figure 4

The clinical appearance of the patient 12 months following homograft implantation.

Homologous bone transplants have their greatest application when segmental losses of bone occur, either from traumatic cause or locally destructive neoplasia. Such lesions as giant cell tumor of bone, central chondrosarcoma, parosteal sarcoma, aneurysmal bone cyst, fibrous dysplasia and low grade cell types of fibrosarcoma are conditions in which homograft transplant should be considered.[24,28] Although transplantation of entire joint homografts have been performed, the long term results are generally disappointing, especially in weight bearing joints.[19,20,22,24,28,29] Degenerative changes to the transplanted joint surface follow and if ligamentous structures are included in the transplanted material a loss of proprioceptive nerve endings leads to stretching of ligamentous structures with eventual joint instability. Thus the ideal application of homograft transplants is in the segmental replacement of skeletal bone without the need for the replacement of the joint surface, especially weight bearing, where postoperative motion will be permitted.

Patients who present with lesions with metastatic potential and who are considered candidates for homologous transplants must be thoroughly evaluated to rule out the presence of tumor activity beyond the primary site.[24,28] Laminograms of the chest are mandatory, as small isolated lesions can easily be overlooked with routine chest x-rays, especially in the retrocardiac area. The selection of patients to receive bone transplants must be carefully arrived at, as the slow process of host substitution of the graft makes protection from excessive stresses and longterm bracing an essential. In some patients, amputation with endoprosthetic replacement may offer a more functional result, achievable over a shorter postoperative time period.

Several technical considerations should also be mentioned. In the selection of homograft bone, infectious or osteopenic donors should be excluded; epiphyseal closure should be complete.[14] Freeze dried and frozen specimens are comparable in regard to host acceptance and have been shown to be significantly better than other types of preparations. Freezing at -20°C. has been shown to decrease antigenicity and improve the biological function of the homograft.[2,8,12,13,14] Harvest of the graft should be performed under ideal sterile conditions. The graft should be placed in an antibiotic solution at the time of harvest and at the time of thawing. Fixation of the homograft should be as efficient as possible due to the prolonged period of healing. Intramedullary fixation with a step cut, dovetailed configuration at either end of the graft is generally most ideal.[14,24,25]

Summary: The use of homologous bone transplants in the treatment of large locally destructive bone lesions is indicated in certain selected situations. The patient must not exhibit evidence of metastatic spread of the tumor. The patient must be willing to accept a prolonged period of guarded activity necessary for incorporation of the graft by the host. En bloc resection with prosthetic replacement must be ruled out as mechanically unsuitable. Sound mechanical fixation, preferably by intramedullary means, is mandatory. Transplantation of homologous joint surfaces, especially in weight bearing joints, generally does not provide for a durable or satisfactory joint surfaces, especially in weight bearing joints, generally does not provide for a durable or satisfactory joint surface. Good to excellent results are achievable if the principles of bone homograft transplantation are carefully observed.

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THE STAGES OF GRIEF EXPERIENCED BY PARENTS OF HANDICAPPED CHILDREN

MARGARET S. TENBRINCK, M.D., FAAP
PAUL W. BREWER, PH.D.

Awareness of these stages of reaction should be shared with the parents of handicapped children. Silence on the part of relatives and friends is not as helpful as an open show of interest and care and especially a sincere offer to help.

Parents of exceptional children learn of their children's handicaps at different times and in different ways.

A large group of these children is identified at birth. Some are obvious, such as birth defects like meningomyelocele. Down's syndrome is only rarely missed by alert hospital personnel. The premature and the low Apgar score babies are immediately tagged as infants "at risk."

Submitted for publication 5/76.
From: Child Evaluation Center, Phoenix, AZ. (Dr. Tenbrinck, Retired Director; Dr. Brewer, Former Chief Psychologist. Reprint requests to Dr. Brewer, 1313 N. 2nd St., Phoenix, AZ 85004.

Another group of children slip by in their first year as "quiet and good babies", only to come to be noticed later because of their delayed psychomotor development, usually in comparison with other children in the environment.

Parents may ignore a delay in walking and talking in the toddler only to come to grips with the problem when they realize their child will not be able to compete in a peer group, such as preschool or kindergarten.

Another group of borderline retarded children only surfaces when the child is challenged by the academic demands of our school system.

Regardless of the age of the child, the severity of his handicaps, and the manner in which the parents become aware of his abnormality, the recognition and awareness of their child's condition come as a great shock. The stages of parental reactions are, in many ways, similar to those experienced by the families of the dying as described by E. Kubler-Ross.*

Following the awareness of the child's condition and the shock this causes them, is a period of denial and isolation on the part of the parents. They are unable to face the facts of their child's handicaps and, instead, start to shop around for more optimistic opinions. There may be many reevaluations. This period serves as a buffer zone after the unexpected shocking news and allows time for them to collect and mobilize less radical defenses. This stage is usually temporary.

The second stage is explosive and characterized by anger, rage, envy, and resentment. They are angry at people and at God. "My God, My God why has Thou forsaken me?" (Psalm 22:1, Matthew 27:48, Mark 15:34). Their anger becomes projected towards those involved, such as "Doctors are no good." Their grievances cause them to complain and make demands. At the same time, some hope now begins to manifest itself.

Long before the anger and aggressions are dissipated, a long period of bargaining is overlayed. This is the time of attempting magic cures supported by recovery fantasies. We have seen some parents pursue a series of quackeries until the end of their child's adolescence. Their behavior represents a sort of agreement to postpone the inevitable. During this combined period there may also be a short period of partial denial. Parental attitudes and promises may be associated with quiet guilt.

In the fourth stage stoicism and rage become replaced with a sense of great loss and depression. Financial burdens are often added at this time. A handicapped child needs specialist care, operations, special equipment, and special schooling. Necessities preclude small luxuries. Many dreams will no longer come true. These depressions may be reactive, in which case encouragement and reassurance are meaningful. Or they may be preparatory for complete acceptance of the inevitable.

The fifth stage is acceptance and is characterized by realistic coping. When the parents arrive at this stage they are ready to plan unemotionally

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*Kubler-Ross, Elizabeth: On Death and Dying. The Macmillan Company, New York, N. Y., 1969.

STAGES OF GRIEF
EXPERIENCED BY PARENTS OF HANDICAPPED CHILDREN

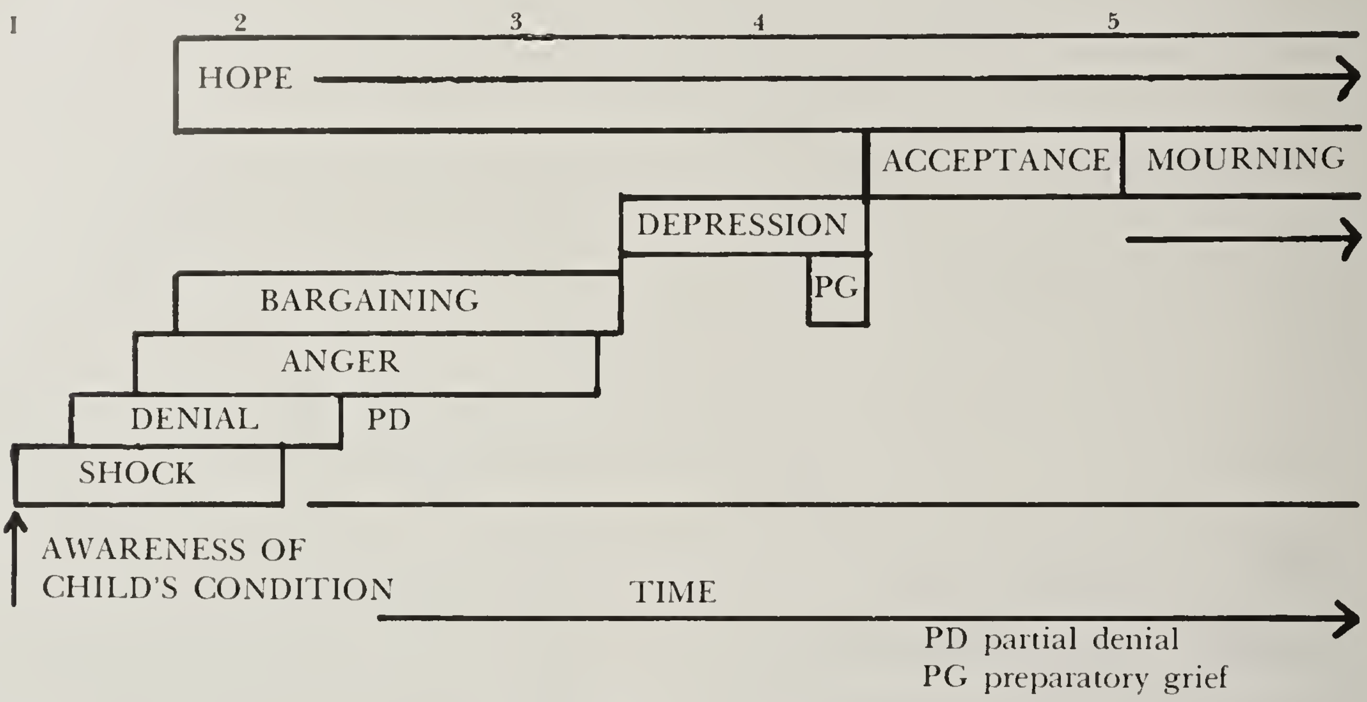

Table 1

for the best possible future of their child. (Table 1)

With the awareness of mental retardation the loss reaction or grief does not end quickly as it does with the finality of death. Instead there is unending grief and chronic sorrow. The stages described are defense mechanisms enabling parents to cope with their grief. The stages last for different lengths of times, they replace each other or exist at times side by side. Throughout there must be hope. Life must have some meaning.

The role of the family in caring for a retarded child is of utmost significance and will contribute to his response. There may be changes in the household and subsequent effects on the family. Chores may multiply and financial security may diminish. The household should not be totally disrupted. Families need occasional change and should continue to handle their energies economically. It is important that no one, especially the mother, exert herself to the point of collapse.

Problems of communication should not be neglected. Relatives, especially grandparents, have to work through their feelings of grief and guilt. They should not be blamed for daring to ventilate socially poorly tolerated thoughts of anger and resentment. Prolonging grief, shame, and guilt results in physical and emotional ill health.

The resolution of grief and anger is followed by the long period of mourning. If their anger has been tolerated, whether directed at the physician, God, or someone else, then the family will be helped through toward acceptance without guilt.

In interviewing the parents of the children we have evaluated for possible mental retardation and other handicaps, we have found the appreciation of these stages of grief very helpful in understanding parental reactions. Often, in the course of the long evaluations, the parents are enabled to pass through several of the stages. At the first interview, usually the social study, they often deny any problems. In one (unpublished) study we did of 200 reevaluated children, 73% of the parents accepted the findings by the end of the evaluation at the parent conference when the child's future was discussed and planned. We have on occasion, on the other hand, had parents continue to deny their child's handicaps despite the many examinations completed during the evaluation. Saddest to us are the parents who seek quackery as a magic cure.

We are reporting this adaptation of Kubler-Ross's analysis of the stages of dying suggesting it may aid other physicians and allied professionals in dealing with the chronic sorrow and unending grief of parents of handicapped children.

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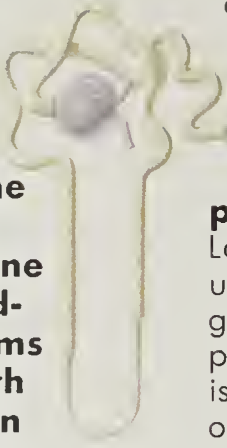

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(See next page for prescribing information.)

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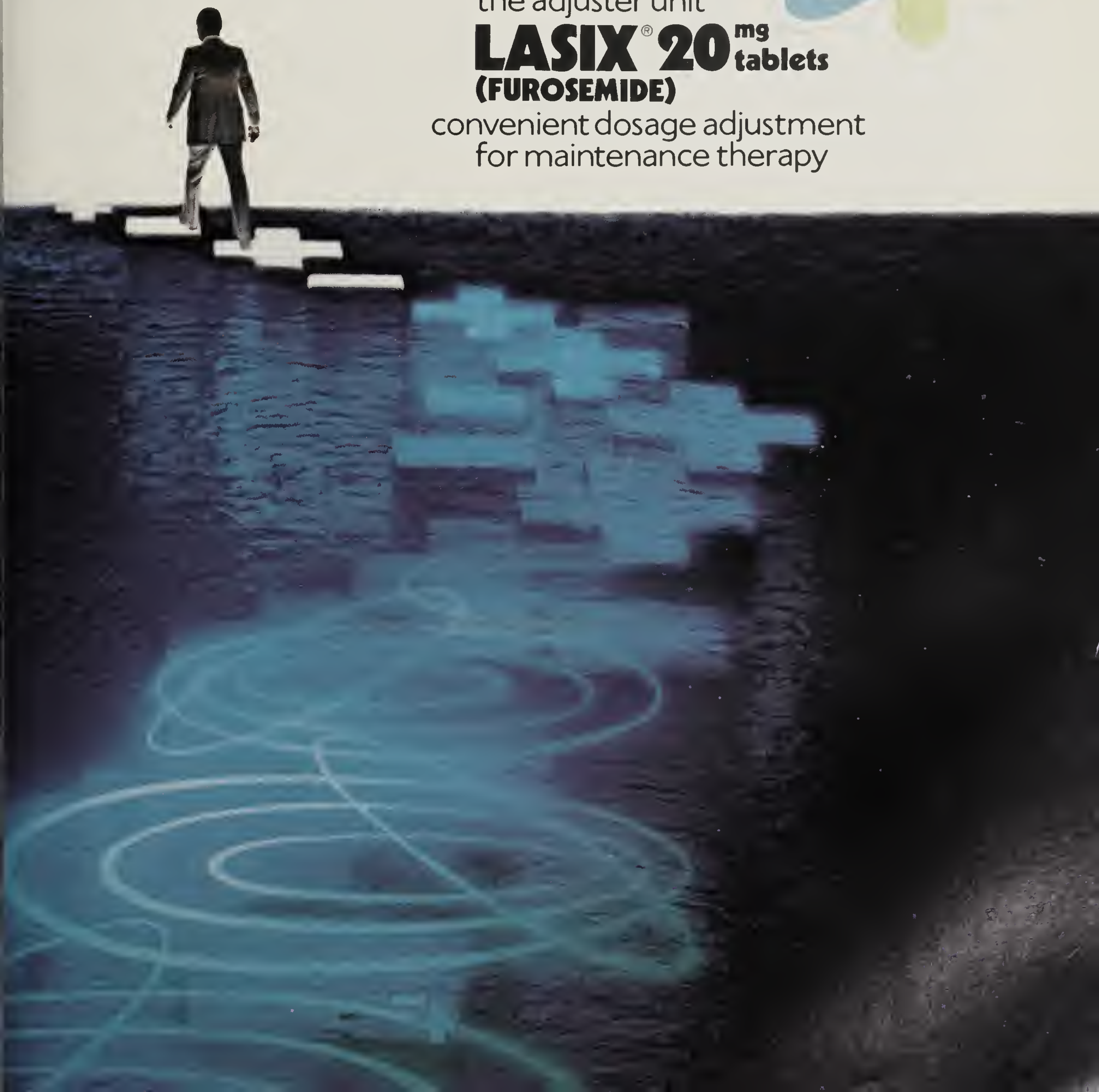

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Indications—Lasix (furosemide) is indicated for the treatment of the edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Hypertension—Lasix (furosemide) may be used for the treatment of hypertension alone or in combination with other antihypertensive drugs. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controllable with Lasix (furosemide) alone.

CONTRAINDICATIONS—Because animal reproductive studies have shown that Lasix (furosemide) may cause fetal abnormalities, the drug is contraindicated in women of childbearing potential. (See "Additional Information.")

Lasix (furosemide) is contraindicated in anuria. If increasing azotemia and oliguria occur during treatment of severe progressive renal disease, the drug should be discontinued. In hepatic coma and in states of electrolyte depletion, therapy should not be instituted until the basic condition is improved or corrected. Lasix (furosemide) is contraindicated in patients with a history of hypersensitivity to this compound.

Warnings—Excessive diuresis may result in dehydration and reduction in blood volume, with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients. Excessive loss of potassium in patients receiving digitalis glycosides may precipitate digitalis toxicity. Care should also be exercised in patients receiving potassium depleting steroids.

Frequent serum electrolyte, CO_2 and BUN determinations should be performed during the first few months of therapy and periodically thereafter, and abnormalities corrected or the drug temporarily withdrawn.

In patients with hepatic cirrhosis and ascites, initiation of therapy with Lasix (furosemide) is best carried out in the hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma; therefore, strict observation is necessary during the period of diuresis. Supplemental potassium chloride and, if required, an aldosterone antagonist are helpful in preventing hypokalemia and metabolic alkalosis.

Patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

In those instances where potassium supplementation is required, an oral liquid preparation should be used rather than enteric-coated potassium salts.

There have been several reports, published and unpublished, concerning nonspecific small-bowel lesions consisting of stenosis, with or without ulceration, associated with the administration of enteric-coated thiazides with potassium salts. These lesions may occur with enteric-coated potassium tablets alone or when they are used with nonenteric-coated thiazides, or certain other oral diuretics.

These small-bowel lesions have caused obstruction, hemorrhage, and perforation. Surgery was frequently required, and deaths have occurred.

Available information tends to implicate enteric-coated potassium salts, although lesions of this type also occur spontaneously. Therefore, coated potassium-containing formulations should be administered only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Patients with known sulfonamide sensitivity may show allergic reactions to Lasix (furosemide).

Precautions—As with any potent diuretic, electrolyte depletion may occur during therapy with Lasix (furosemide), especially in patients receiving higher doses and a restricted salt intake. Electrolyte depletion may manifest itself by weakness, dizziness, lethargy, leg cramps, anorexia, vomiting, and/or mental confusion.

Asymptomatic hyperuricemia can occur and gout may rarely be precipitated. Reversible elevations of BUN may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

When parenteral use of Lasix (furosemide) precedes its oral use, it should be kept in mind that cases of tinnitus and reversible hearing impairment have been reported. There have also been some reports of cases in which irreversible hearing impairment occurred. Usually, ototoxicity has been reported when Lasix (furosemide) was injected rapidly in patients with severe impairment of renal function at doses exceeding several times the usual recommended dose and in whom other drugs known to be ototoxic were often given. If the physician elects to use high dose parenteral therapy in patients with severely impaired renal function, controlled intravenous infusion is advisable (for adults, it has been reported that an infusion rate not exceeding 4 mg Lasix [furosemide] per minute has been used).

Increases in blood glucose, and alterations in glucose tolerance tests with abnormalities of the fasting and two-hour postprandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Lasix (furosemide) may lower serum calcium levels, and rare cases of tetany have been reported.

Patients receiving high doses of salicylates, in conjunction with Lasix (furosemide) may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Diuretics such as furosemide may enhance the nephrotoxicity of cephaloridine. Therefore, Lasix (furosemide) and cephaloridine should not be administered simultaneously.

Sulfonamide diuretics have been reported to decrease arterial responsiveness to pressor amines and to enhance the effect of tubocurarine. Great caution should be exercised in administering curare or its derivatives to patients undergoing therapy with Lasix (furosemide), and it is advisable to discontinue Lasix (furosemide) for one week prior to any elective surgery.

Adverse Reactions—Various forms of dermatitis, including urticaria and rare forms of exfoliative dermatitis, erythema multiforme, pruritus paresthesia, blurring of vision, postural hypotension, nausea, vomiting, or diarrhea.

Anemia, leukopenia, aplastic anemia, and thrombocytopenia (with purpura). Rare cases of agranulocytosis which responded to treatment.

In addition, the following rare adverse reactions have been reported however, relationship to the drug has not been established with certainty sweet taste, oral and gastric burning, paradoxical swelling, headache jaundice, thrombophlebitis and emboli and acute pancreatitis.

Lasix (furosemide)-induced diuresis may be accompanied by weakness, fatigue, lightheadedness or dizziness, muscle cramps, thirst, increased perspiration, urinary bladder spasm, and symptoms of urinary frequency.

Dosage and Administration

ADULTS

The usual adult dose of Lasix (furosemide) is 20 to 80 mg given as a single dose.

If the diuretic response with a single dose of 20 to 80 mg is not satisfactory, the following schedule should be used: Increase this dose by increments of 20 or 40 mg not sooner than 6 to 8 hours after the previous dose until the desired diuretic effect has been obtained. This individually determined single dose should then be given once or twice daily. The dose of Lasix (furosemide) may be carefully titrated up to 600 mg per day in those patients with severe clinical edematous states.

With doses exceeding 80 mg/day and given for prolonged periods careful clinical and laboratory observations are particularly advisable.

Hypertension—The usual dose of Lasix (furosemide) is 40 mg twice daily both for initiation of therapy and for maintenance. Careful observations for changes in blood pressure must be made when this compound is used with other antihypertensive drugs, especially during initial therapy The dosage of other agents must be reduced by at least 50 percent as soon as Lasix (furosemide) is added to the regimen to prevent excessive drop in blood pressure. As the blood pressure falls under the potentiating effect of Lasix (furosemide), a further reduction in dosage, or even discontinuation, of other antihypertensive drugs may be necessary. It is furthe recommended, if 40 mg twice daily does not lead to a clinically satisfactory response, to add other hypotensive agents, e.g., reserpine, rather than to increase the dose of Lasix (furosemide).

INFANTS AND CHILDREN

Pediatric Administration: The usual initial dose of oral Lasix in infants and children is 2 mg/kg body weight, given as a single dose. If the diuretic response is not satisfactory after the initial dose, dosage may be increased by 1 or 2 mg/kg not sooner than 6 to 8 hours after the previous dose Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

How Supplied—Lasix Tablets 40 mg (furosemide) supplied as white round, monogrammed, scored tablets.

Lasix Tablets 20 mg (furosemide) supplied as white, oval, monogrammed tablets.

Note: Dispense in dark containers. Exposure to light may cause sligh discoloration which, however, does not alter potency.

Additional Information

Toxicology

The acute toxicity of Lasix (furosemide) has been determined in mice rats, and dogs. In all three animal species, the oral LD_{50} of Lasix (furosemide) exceeded 1000 mg/kg of body weight, while the intravenous LD_5 ranged from 300 to 680 mg/kg. Intragastric injection of the drug in newborn rats resulted in an LD_{50} of 380 mg/kg.

The acute toxicity of high doses of Lasix (furosemide) was characterized by convulsions, paralysis, and collapse. Surviving animals often became dehydrated and depleted of electrolytes due to the diuresis induced by Lasix (furosemide). In the newborn rats, intragastric injection of the drug caused hyperactivity and anorexia.

Chronic toxicity studies with Lasix (furosemide) were done in rat and dogs. In a one-year study in rats, renal tubular degeneration occurred with all doses higher than 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day). A six-month study in dogs revealed ca cification and scarring of the renal parenchyma at all doses above 1 mg/kg (83 percent of the maximal recommended human dose of 600 m per day).

Reproductive Studies

The effects of Lasix (furosemide) on embryonic and fetal development and on pregnant dams were studied in mice, rats, and rabbits.

Lasix (furosemide) caused unexplained maternal deaths and abortions in the rabbit when 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day) was administered between days 12 to 1 of gestation. In a previous study the lowest dose of only 25 mg/kg (2 time the maximal recommended human dose of 600 mg per day) caused maternal deaths and abortions. In a third study, none of the pregnant rabbit survived a dose of 100 mg/kg. Data from the above studies indicate feta lethality which can precede maternal deaths.

The results of the mouse study and one of the three rabbit studie also showed an increased incidence of hydronephrosis (distention of th renal pelvis and, in some cases, of the ureters) in fetuses derived from treated dams as compared to the incidence in fetuses from the contro group.

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THE COCONINO MEDICAL CAPER

JOHN W. KENNEDY, M.D.

The Coconino Mountain men have done it again and pulled off a great symposium. The weather was superb, cool with a shower and a bit of hail each evening. The galaxy of professorial speaking stars regaled the knowledge seekers with great stores of medical information and wit.

This was the 12th year of this mid-summer symposium and it has become so popular that registration in future years, will probably be limited to two hundred enrollment.

As a tone deaf country music boy, I am hardly the one to become ecstatic about concert pianists and symphony musical notes, but I am reliably informed and indeed I must agree, that the Festival Concert was superb.

Dr. Louis Limberg of Miami University School of Medicine held forth on heart disease and put to rest the old fable that "loading doses" of digitalis were needed. Simply start out with the daily dose and go from there, for a non-cardiologist this seemed blasphemy as judged from my teachings in medical school lo these many morns ago.

Dr. G. Hugh Lawrence of the University of Oregon School of Medicine blew away the efficacy of radical lung surgery in carcinoma of the lung. It doesn't increase the longevity. And in that respect, perhaps the surgeons have arrived at the same conclusion in this organ malignancy as in breast carcinoma, so-called radical surgery holds little promise. But there is some promise in using the new immune reaction enhancing agents.

Dr. Richard Hamilton of Stanford University, Department of Neurosurgery held forth on head injuries and one of his axioms was - admit anyone who has been unconscious and can't remember the accident. These people are prone to develop symptoms later, many times that of epidural hematoma. He also recommended that the cervical spine be X-rayed with every skull injury.

And then we had that famous Irish wit, Andrew Welton of "Jim Hiskins" of Baltimore, sometimes known as the Johns Hopkins who regaled us with a wide display of erudition concerning the kidney. Of little practical medical value, but of some social economic consequence is the fact that it is cheaper to keep a patient on dialysis a year than to keep a prisoner confined in jail for a year. There must be a lesson here, but I am not sure what it is.

These gems are not at all fair to the speakers, their presentations were practical, well spoken, and aptly illustrated.

We therefore join all the students at this symposium in congratulating Dr. Fredell and his committee on the careful planning, precise execution, and salubrious weather which they supplied with the program. It will be hard for them to top this performance on the 12th of the series, but no doubt they will do it and it will happen next year.

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Dean's Page

THE PAIN CLINIC AT THE COLLEGE OF MEDICINE

NEAL A. VANSELOW, M.D.

Effective therapy of chronic, intractable pain syndromes is one of the least understood subjects of medicine. Philosophically, the practitioner has been inculcated with the concept, correct in the main, that pain is but a symptom of an underlying pathologic process. Except for the ephemereal use of analgesic medications, the major task of the physician is to diagnose the cause of the pain, and specifically treat the pathology. In essence pain as pain is ignored. Few medical schools teach students rational management of a symptom such as pain. After all, pain is intangible. It cannot be measured, x-rayed, dissected, or tested. Because no animal model mimics the clinical situation, laboratory simulation is difficult. Acute pain produced in healthy volunteers does not parallel chronic pain with its attendant psychologic and economic problems. It is now well recognized that given six months duration of intractable pain, personality changes are inevitable. Standard psychologic tests, normal prior to the onset of pain, change. One can see how easy it is for the physician to escape a difficult situation by declaring that the patient's pain is either imagined or due to neurosis.

The Pain Clinic at the College of Medicine was inaugurated in early 1972 with the objectives of rendering service, teaching, and research in the area of conservative management of intractable pain syndromes. Over 3,500 patient visits have occurred to date. Therapeutic modalities employed include pharmacologic regimens, physical therapy, minor and major nerve blocking, and psychologic counseling. Three investigative protocols have been completed: 1) evaluation of acupuncture in chronic pain syndromes; 2) use of a new myolissive compound, cyclobenzaprine, in chronic low back and cervical pain; 3) employment of a new topical salicylate derivative in low back pain. But most important many undergraduates and residents have now been exposed to clinical intractable pain syndromes in the Pain Clinic, and taught that there can be effective therapy for this public health problem, even though at times that therapy is supportive. Very few medical schools in the United States can say as much.

Neal A. Vanselow

Clinical Oncology
In Arizona

COMPUTED TOMOGRAPHIC SCANNING

ROBERT S. WALDMAN, M. D.
DONALD A. TOBIAS, M. D.

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INTRODUCTION

Computed tomography represents a significant new development in diagnostic radiologic imaging. Although the concept of image reconstruction is not new, it remained for Godfrey Hounsfield and a group of engineers working at E.M.I. Ltd. to develop the first computerized axial tomographic scanner.[1,2] The initial scans were crude compared to those now available, but it was obvious that a major breakthrough in diagnostic imaging had occurred. The prototype unit, and those first commercially available, were designed for scans of the head. It became evident as clinical experience increased that diagnoses of a host of pathological processes within the brain, including neoplasm, vascular abnormalities, intra- and extracerebral hemorrhage, inflammatory processes, etc., as well as evaluation of the ventricles and subarachnoid space could be easily made by this non-invasive technique.[3,4]

Technological breakthroughs were made permitting greater definition in the scans, as well as the development of a whole body computerized tomographic scanner. With the development of the whole body scanner[5,6], computed tomographic scanning will have a significant impact in the field of oncology, and this is the subject of our paper.

TECHNOLOGY

The history and development of computerized axial tomographic scanning, as well as the detailed technology is well reviewed in several excellent articles.[1,2,6,8] Computed tomographic (CT) scanning represents the marriage of an X-ray imaging device and the computer. The system consists of an X-ray source, which is coupled to a detector system, generally a sodium iodide crystal. Newer, more sensitive detector systems are being developed. The X-ray photons pass from the source through the area being examined and then strike the detector. If there is a great deal of attenuation of the X-ray beam, then the detector records only a small amount of X-ray flux. Conversely, if there is little attenuation then a great deal of X-ray flux is recorded. The source-detector system moves linearly and then rotates through a 180 degree arc to produce a cross-sectional image. The information from the detector is being fed constantly into a computer which has been programmed to solve a complex series of equations or algorithms and to plot the X-ray absorption values on a matrix. The original matrix was 80x80 units, and can be thought of as a piece of graph paper with 80 boxes in both the X and Y axes. The computer then assigns to each box within this large piece of "graph paper" an average value of X-ray absorption. As technology became more sophisticated, the matrix became finer, producing

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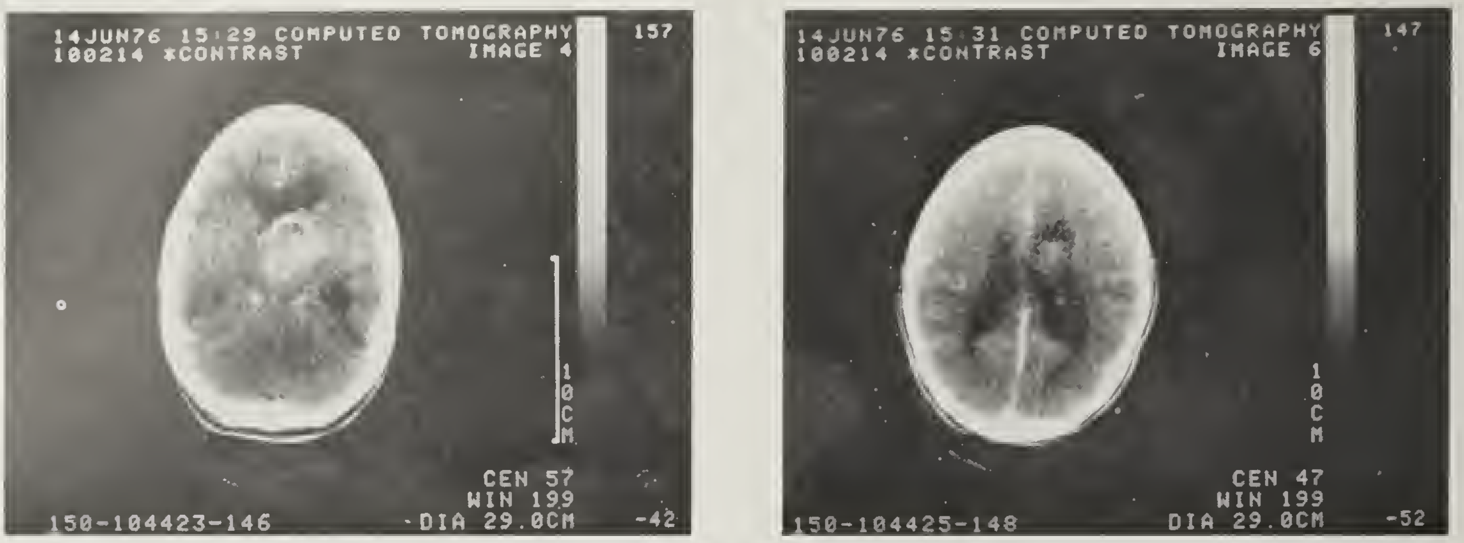
Reprint request to: Computed Tomography, Ltd., 1728 West Glendale Avenue, Suite LL3, Phoenix, Arizona 85021.



1A

1B

Figure 1

Large glioblastoma in the right thalamic region, 1A extending into the right lateral ventricle 1B. There is displacement of midline structures and ventricular dilatation. Contrast enhancement was employed.

images with greater detail and quality. The same basic technology was used to build the first whole body scanner. The field of computed tomographic scanning is advancing at an exceedingly rapid pace with development of scanners capable of producing images more quickly and with greater clarity.[9]

CLINICAL APPLICATIONS

Computed tomographic scanning will have the greatest impact on oncology in four primary areas (Table 1). The first is that of tumor diagnosis and determination of local extension of tumor. The second is for evaluation of metastatic disease. We believe that CT scanning may well replace radionuclide scanning for evaluation of metastatic disease particularly in the liver and brain. Third, CT scanning will be invaluable to follow quantitative tumor response to various modes of therapy. Fourth, the cross-sectional CT scan may be utilized, in conjunction with computerized therapy planning modes, to produce extremely accurate radiation therapy plans. In addition to defining the extent of the tumor within the precise body contour, computed tomography will provide information on the X-ray absorption of the normal tissue through which the beam passes. Using this information in a computerized therapy planning system, maximum tumor dose can be accomplished with minimal damage to normal surrounding tissue, allowing for the most effective radiation therapy program. We will explore how each of these will be important for various tumors in different regions of the body:

Table 1

Applications of CT Scanning in Oncology

1. Tumor diagnosis
2. Staging for extent of disease
3. Search for metastatic disease
4. Assessing response to therapy
5. Radiation therapy planning

I. Head and Neck:

Computed tomography has established itself in nurmerous reports,[3,4,7] as an excellent noninvasive technique for the diagnosis of primary (Figure 1) and metastatic (Figure 2) intracranial lesions. The exact location and size of such lesions can be determined. Response to surgery,

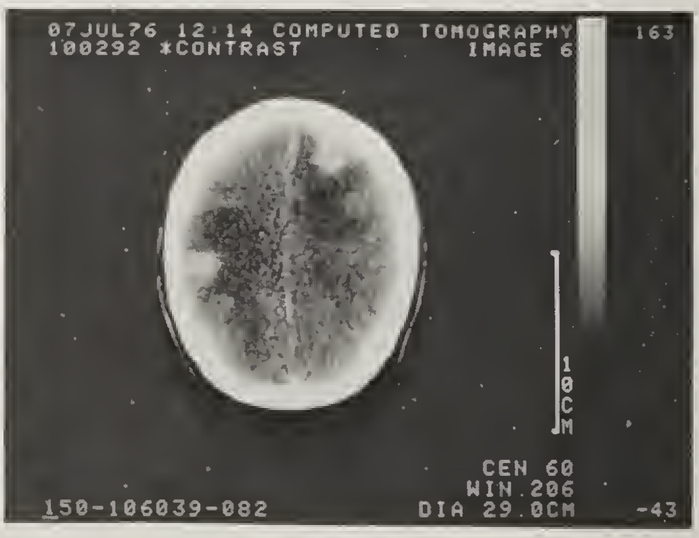


Figure 2

Multiple areas of increased density following contrast enhancement represent metastatic foci in the brain in a patient with primary carcinoma of the lung.

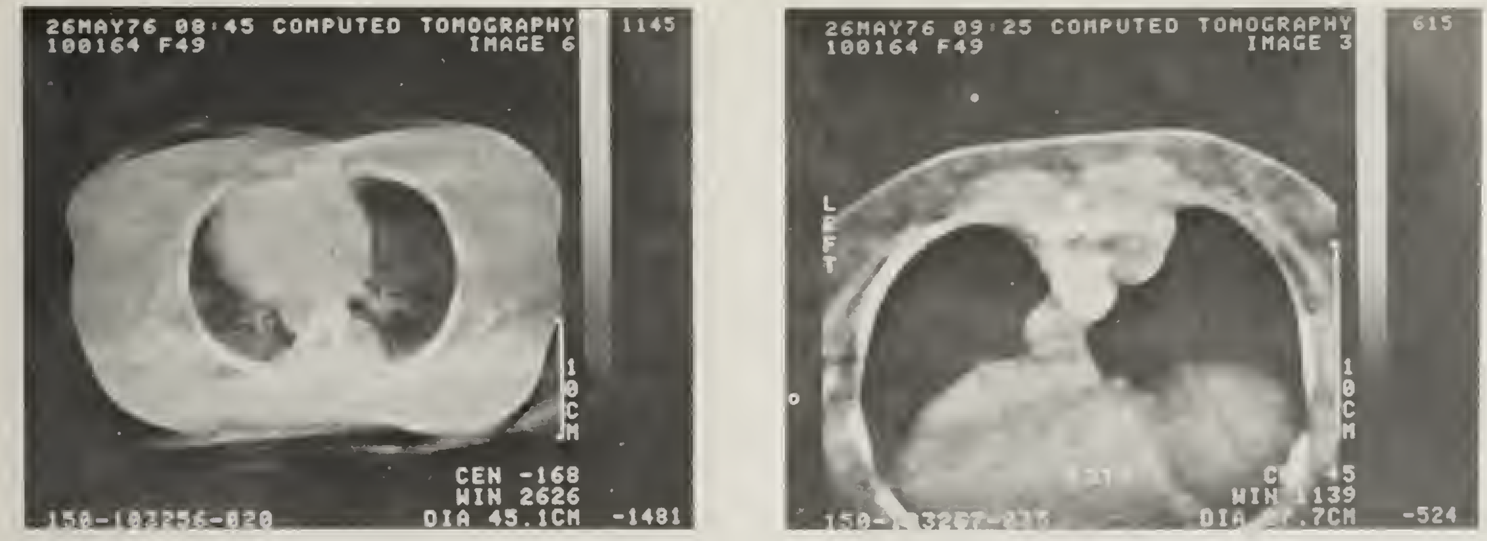


Figure 3

3A

3B

(3A) Supine CT scan demonstrates a large lesion located posteriorly in the right paraspinous region. (3B) Prone magnification view shows the lesion widening the neural foramen. Diagnosis: "dumbbell" neurofibroma.

chemotherapy or radiation therapy can be monitored by follow-up scans. In addition, soft tissue lesions in the orbit, nasopharynx, oropharynx, paranasal sinuses, and cervical area with local extension and bone destruction are well seen. Computed tomography has demonstrated its effectiveness in differentiating small gradations of X-ray soft tissue absorption, with obliteration of fine soft tissue fascial planes, as well as subtle changes in the thin, fine bony structures of the head and neck, allowing for detection of even small, occult lesions. Precise therapy planning in this difficult region is made easier with the use of cross-sectional CT scans.

II. Chest:

Conventional chest films and tomography will remain the primary tools in radiological evaluation of pulmonary neoplasm. Computed tomography will be a valuable adjunct in those areas which are not well seen on conventional films.[9] The mediastinum is an excellent example of how anatomic detail can be studied, taking advantage of subtle differences in X-ray absorption. Normal lymph nodes can be separated from one another by fine fat layers in the fascial planes. Obliteration of these normal fat planes is seen when the nodes become involved with carcinoma or lymphoma. These subtle changes in the fatty fascial planes are not evident on conventional films. The retrocardiac area and lung apices can be seen to best advantage with cross-sectional CT scans. The sensitivity of computed tomography allows for detection of calcification within nodules not seen on conventional tomography. This can be extremely helpful in differentiating between benign and malignant disease. Intra- and extra-pulmonary lesions (Figure 3) can be distinguished on the cross-sectional scans and very small amounts of pleural effusion are readily detected.

Response to therapy in mediastinal lymphomas can be monitored with serial CT scans. Any change in the status of this disease can be quickly determined quantitatively to enable appropriate changes in therapy. In radiation therapy planning with determination of the X-ray absorption value of the lung by CT scanning, a more optimal radiation therapy plan can be initiated. This will provide maximum tumor response with minimal damage to the adjacent normal lung tissue, particularly helpful in the elderly or those with chronic lung disease where pulmonary reserve is limited.

III. Abdomen and Pelvis:

The liver, pancreas, and retroperitoneal structures—areas not readily accessible except by means of invasive diagnostic x-ray techniques—are well visualized by computed tomography.[9,10]

Primary and metastatic liver neoplasms are well demonstrated on contrast enhanced CT scans. Although preliminary reports in the literature indicated equal accuracy in detecting liver metastases when comparing CT and radionuclide scanning, with increasing clinical CT experience, and the advent of fast scanners providing greater definition, it is obvious that

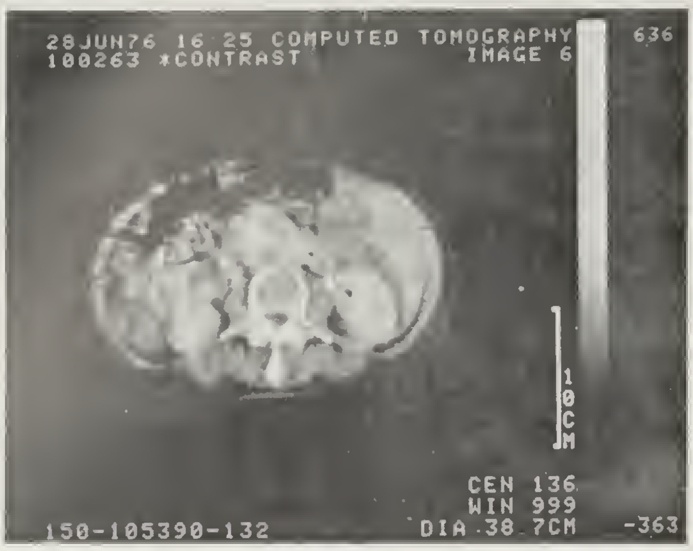


Figure 4

Carcinoma in the head of the pancreas with secondary enlargement of the gallbladder and biliary tract.

CT scanning will surpass radionuclide scanning in the evaluation of both primary and metastatic neoplasms of the liver. In patients with jaundice, CT scans will differentiate the obstructive from the non-obstructive type so that the appropriate therapy can be initiated.

Evaluation of pancreatic neoplasms is difficult even with invasive angiographic techniques. With contrast enhancement to outline normal vascular landmarks and a small amount of ingested Gastrografin to visualize the upper gastrointestinal tract, the anatomy of the pancreas can be well delineated by CT scanning. Alterations in pancreatic morphology associated with secondary changes in surrounding structures has been reported with pancreatic carcinoma (Figure 4), pancreatitis and pseudocyst.[5,9,10]

Abdominal lymphoma with retroperitoneal and para-aortic lymph node involvement is well demonstrated on CT scans. In addition, this scanning mode offers a faster, less traumatic method than lymphangiography in evaluating abdominal node involvement. Quantitative changes in the lymph nodes following chemotherapy or radiation therapy can be easily evaluated by the use of serial, non-invasive CT techniques.

Renal neoplasms and cysts can be differentiated on CT scanning, particularly following contrast enhancement. With contrast enhancement the renal parenchyma shows the greatest increase in X-ray absorption. Cysts show virtually no increase in X-ray absorption following contrast enhancement. Tumors fall in the intermediate range. Benign asymptomatic renal cysts are a common finding on abdominal scans.

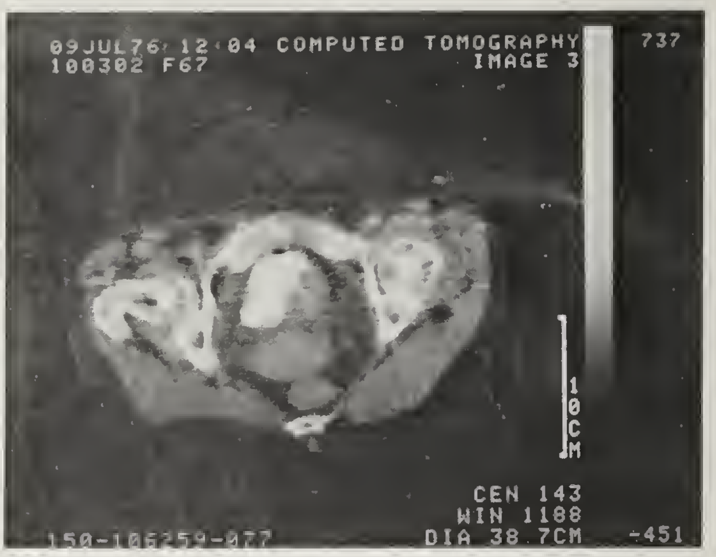


Figure 5

Carcinoma in the right wall of the bladder following contrast enhancement. The scan demonstrates that the tumor is limited to the bladder and the right lateral pelvic wall is free of disease.

Adnexal, uterine, bladder and prostate neoplasms are also well demonstrated by computed tomography. CT scanning permits precise determination of the lateral, anterior-posterior, as well as supero-inferior extension of the disease process, allowing for accurate staging of these neoplastic processes. For example, in a bladder carcinoma (Figure 5), one can determine whether there is extension beyond the wall and the degree of this extension. This allows for optimal therapy planning.

In summary, computed tomography is a milestone technological advance in radiological imaging that is producing a significant impact on oncology today and will have an even greater impact in the future.

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Obstetrics and Gynecology

SOAP

DONALD J. ZIEHM, M.D., EDITOR

The progress of medicine can probably be measured by the number of specialty societies in any given area of practice. As subspecialization develops within a discipline, investigators and clinicians will eventually band together to form a "society." Serving the purposes of sharing advances in technology and treatment among its members, and providing valuable continuing education to practitioners, most "societies" are an integral part of medicine.

With the development and growth of Perinatology and its allied fields it was inevitable that a "society" should be spawned. The Society for Obstetric Anesthesia and Perinatology (SOAP) is little known in the Western United States. Its greatest popularity has occurred in the Northeast and Southeastern areas. It is the intention of the editor that the existence of this organization not go unrecognized. In order to acquaint Arizona physicians with SOAP, its history and purposes, two abstractions of previously published articles are presented. The advise, encouragement, and assistance of Dr. G. Ostheimer, editor of the SOAP Newsletter and member of the Department of Anesthesia at the Boston Hospital For Women is also acknowledged. It is our sincere hope that many inquiries concerning membership in this very worthwhile organization will be forthcoming from physicians and paramedical personnel with a strong interest in Perinatal Medicine.

Editor

With the roster of obstetric anesthesiologists steadily growing, the time was ripe for an informal society which would foster discussions of common problems with knowledgeable and interested obstetricians and pediatricians. Credit for getting such an organization off the ground must go to R. F. Hustead, Phillips, and Smith. The first meeting took place in Kansas City in the fall of 1969. More than 50 physicians with special interest in maternal and/or neonatal well-being attended and chose to name the new organization the "Society for Obstetric Anesthesia and Perinatology" (SOAP). The second annual meeting, held in Nashville in the spring of 1970, attracted nearly twice the original number of participants. The Society was formally organized at its third meeting (Atlanta, 1971). The Articles of Organization and By-Laws state that "The Society does not seek corporate status nor legal identity . . . The purpose of the organization is to provide a forum for discussion of problems unique to the peripartum period. This includes clinical practice of medicine, basic research, practical business and public health aspects of this important phase of life . . . This Society is dedicated to informality, and to effective facilitation of the unrestricted and spontaneous interchange of new ideas in this field. It further seeks to disseminate pertinent new information to other medical and lay groups and to stimulate improved practices in all aspects of this field . . . Any physician or scientist particularly interested in the problems of the perinatal period may become a member of the organization. Others may attend the annual meetings as guests . . . There shall be only one class of members: Active Membership . . . The Annual Meeting must be held on a weekend."

SOAP has continued to grow. Of 130 registrants at our sixth meeting (San Francisco, 1974), 77 were anesthesiologists, 22 pediatricians, 19 obstetricians, and 12 in other disciplines. From the onset, SOAP has published a quarterly Newsletter, edited by Shnider during the first four years and by G. W. Ostheimer since. More important, SOAP has initiated two reviews, one on "complications of fetal monitoring" and one on "timing of postpartum tubal ligation." SOAP also has instituted a registry of "blood patch" treatment for post-spinal headache, (the results of which have been published.)

Annual meetings of SOAP have planned scientific sessions. Formal presentations on a specific topic are delivered by guest speakers. Reviews of "what is new in obstetric anesthesia," "what is new in obstetrics," and "what is new in neonatology" are given by members who are specialists in these fields. Presentations of "work-in-progress" are followed by discussions and constructive criticism. All interested physicians are welcome.

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Despite the marked decrease in deliveries in the United States over the past eight years, the interest in obstetric anesthesia and perinatology continues to grow . . . The public and the medical profession have come to the realization that the parturient and the fetus in transition to the newborn deserve the best medical care we can offer as well as our understanding and compassion. We can be proud of the many accomplishments our Society (S.O.A.P.) has made. We have official recognition with ASA and ACOG. We have been asked to actively participate in various functions of both these organizations. It appears that the AAP may wish to formalize relations with us. Our members and our organization have greatly influenced and upgraded maternal and perinatal care. Yet this has not been done by threats and proclamations that cause ill will, but rather through education and advice given when asked. Politics and in-fighting have not occurred in our organization. We can look to the past with a sense of pride and accomplishment.

Yet, we can't rest on laurels if SOAP is to remain a viable and worthwhile organization. Changes will be necessary, but let us hope they will be made to enrich and solidify our principals, which were so wisely incorporated into our constitution.

Let me conclude my term in office with my own personal feelings as to what these principles are and why they should be maintained. (1) Our most important aim is to improve the care in the perinatal period, to make birth for the parents a safe, yet fulfilling and wonderful experience. We also want to give all new parents the most healthy baby possible. In this, our major goal, we also must recognize the need to consider the emotional needs of patients entrusted to our care, realizing each is an individual. We must emphasize not only the science of medicine but the art of medicine so often neglected today. (2) I would hope that we are to remain an informal forum, exchanging information among ourselves to increase our knowledge as well as that of the medical profession. We present this information not for personal gain, but to enrich each other and to gain the wisdom available from our many diverse skills. By this informal exchange, not only do we enrich ourselves, but we also avoid duplication of our work and act not in competition, but in cooperation among ourselves. This cooperation is uncommon in other branches of medicine. (3) While we strive for informality, closeness, and a sense of comradeship, SOAP must always remain an open forum to all those who truly have a genuine interest in the perinatal period. I am proud that we open our meetings to other scientists, nurses, nurse practitioners, CRNAs, and midwives. It concerns me that many subspecialty groups in anesthesia limit the number of members, possibly to increase their power and prestige often at the expense of greater input to their own society. I sincerely hope SOAP never becomes a clique of the "priveleged." By the same token, I would hope we would attract only those with a genuine interest in the perinatal period. I have opposed this Society becoming a place for physicians and others who need credits for licensure, who have little interest in our goals, from getting such credits. There are already enough refresher courses and luxury cruises for this. (4) Finally, I would hope we would continue to offer to the medical profession leadership in this important and often neglected area of medicine. Yet leadership is often equated with power and telling others what they must do if they are to avoid our wrath. Somehow, to my way of thinking, the greatest leaders have been servants—or as stated by a very wise man, "The measure of a man is not the number of servants he has, but the number of people he serves." Let us exert our leadership through our service.

Dr. Brett Gutsche, outgoing President of SOAP. SOAP Newsletter, June 1976.

Seminars in Endocrinology and Metabolism

HYPERTHYROIDISM: THE UP AND DOWN OF IT

by **LAURANCE B. NILSEN, M.D.**

Continuing with the present issue of Arizona Medicine is the series of articles entitled "Seminars in Endocrinology and Metabolism." The purpose of these short review articles is twofold. First, due to the rapid proliferation of new knowledge in the field of endocrinology and the multiple tests available for their evaluation, short, clinically oriented reviews would enable the physician to keep abreast of these newer developments as they relate to their practice. In addition, with great stress being placed on voluntary recertification in many subspecialties, reviews such as they could serve as an authoritative, succinct teaching forum. The editors will endeavor to accomplish these goals by utilizing the talents of practicing physicians as guest contributors to this series. Feedback, both positive and negative, is encouraged in order to help us fulfill these objectives.

MARSHALL B. BLOCK, M.D. Editor

Although the classical description of patients with hyperthyroidism has not changed over the years, recent advances in thyroid hormone physiology have afforded a better understanding of the pathologic mechanisms involved, and provided methods for still earlier diagnosis.

Elevated peripheral levels of thyroid hormone have been the hallmark of the diagnosis of hyperthyroidism for many years. Recently serum thyroxine (T_4) levels have been accurately measured by the techniques of competitive protein binding and radioimmunoassay, and triiodothyronine (T_3) levels by radioimmunoassay. In past years thyroxine had been thought to be the only hormone secreted from the thyroid gland. It is now evident that triiodothyronine is in fact a major thyroid hormone and that a significant quantity is formed in the peripheral tissues by monodeiodination of T_4 to T_3. Thus circulating levels of thyroid hormone are derived not only from the thyroid gland but also from the peripheral conversion of T_4 to T_3.[1] On occasion measurements of both hormones are needed to establish a diagnosis of hyperthyroidism. This becomes increasingly important in the early stages of Graves' disease and in many autonomously functioning thyroid nodules where levels of T_4 may be normal but levels of T_3 elevated. The usually available measurements of T_4, "T_3 resin uptake", and the product of the two values (T_7 or free thyroxine index) do not specifically measure plasma triiodothyronine; thus a specific T_3 radioimmunoassay is needed.

The majority of patients with Graves' disease present with a diffusely enlarged thyroid gland, fine tremor, and accelerated reflexes. Often exophthalmos, extraocular muscle palsies, pretibial myxedema and onycholysis (Plummer's nails) can be appreciated, permitting a relatively easy diagnosis. However patients presenting with unilateral ophthalmopathy, a normal size thyroid gland, and normal plasma T_3 and T_4 levels pose a particularly difficult diagnostic problem. Intracranial and intraorbital disease must first be excluded by means of computerized axial tomography, orbital ultrasonography and other conventional neurodiagnostic studies. Thyroid hormone secretion may remain in the measurements of T_3 by radioimmunoassay and normal suppression of thyroid function during the administration of exogenous thyroid (Werner's Cytomel suppression test). Although thyroid hormone secretion may remain in the normal range in these patients, the thyroid gland is often found to be functioning autonomously, no longer responsive to the normal "feedback inhibition" mediated through the hypothalamus and pituitary gland. Referred to as "euthyroid Graves' disease", this condition represents an early stage in the evolution of the disorder. Exogenously administered thyrotropin releasing hormone (TRH) often fails to stimulate the pituitary to release TSH normally in these patients, indicating that the plasma T_4 and T_3 levels are indeed elevated slightly for that particular patient, although still falling within the normal limits defined for the normal population.[2] Thus the blunted response to administered TRH affords an exquisitely sensitive method for early diagnosis of hyperthyroidism.

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Graves' disease accounts for most cases of hyperthyroidism in the general population today. Its etiology remains uncertain despite advances in the understanding of thyroid physiology. Although cell mediated and immediate immunologic abnormalities are evident in patients with this disorder, the etiological significance of these data remains controversial.[3]

Unlike the diffusely enlarged and hyperfunctioning gland characteristic of Graves' disease, many older patients present with a multinodular goiter containing one or more autonomous hyperfunctioning nodules producing the hyperthroid state (Plummer's disease). Adjacent areas of normally functioning thyroid tissue are suppressed, but are capable of responding to exogenously administered thyroid stimulating hormone (TSH), unlike Graves' disease in which the entire gland is already functioning maximally and is incapable of any further response to administered TSH. Many of these patients come from the goiterous areas of the midwest and have had a multinodular goiter appreciated for many years. In Plummer's disease the signs of hyperthyroidism are subtle and few in number such as gradual weight loss, tremor and personality changes. The syndrome of apathetic thyrotoxicosis is often seen in these patients and is characterized by weight loss, marked asthenia, sinus tachycardia or atrial fibrillation and frequently congestive heart failure.[4] The diagnosis is often not apparent unless considered by the physician. However, the cardiac abnormalities improve markedly once thyrotoxicosis is identified and controlled.

Less common causes of hyperthyroidism include hyperfunctioning solitary thyroid nodules, excessive administration of exogenous thyroid hormone and Hashimoto's thyroiditis. Although chronic thyroiditis typically leads to hypothyroidism, it has occasionally been associated with thyrotoxicosis early in its course.[5,6] Hashimoto's thyroiditis, like Graves' disease, is thought to be an immune disorder, and the two diseases have been described as a continuum with hyperthyroidism at one end of the spectrum and thyroiditis (and ultimately hypothyroidism) at the other. Whether the simultaneous occurrence of Hashimoto's thyroiditis and Graves' disease is coincidental or just a varient of Hashimoto's thyroiditis remains uncertain. Subacute thyroiditis can also give rise to elevated serum levels of thyroid hormone and mild symptoms of hyperthyroidism early in the disorder; however the radioiodine uptake is typically very low in contrast to that seen in Graves' or Plummer's disease.

The preferred treatment of hyperthyroidism depends on the underlying etiology. Since we do not know the cause of Graves' disease our therapy is directed at slowing or ablating thyroid function. The three recommended treatment modalities each have disadvantages. The most noninvasive treatment for Graves' disease include the use of thioureas which block the incorporation of iodine into thyroid hormone and thus decrease the production of T_7 and T_3. However, thyrotoxicosis recurs in up to 40% of patients after discontinuing medication. Radioactive iodine or surgery provide definitive treatment, but hypothyroidism will often develop within five to ten years with either therapy. There has been concern that radioactive iodine treatment in young patients may induce mutagenic changes in the mitotically active thyroid cells leading to thyroid carcinoma after a latent period of 10-20 years.[7] Not all longterm followup studies have substantiated this fear.[8] In fact, I^{131} is considered the treatment of choice for non-pregnant adults with Graves' disease. Nevertheless, subtotal thyroidectomy after appropriate medical preparation has been reserved for thyrotoxic patients with unusually large goiters, for patients under the age of 18, and for many thyrotoxic patients during pregnancy. As the etiology of Graves' disease is understood, more appropriate and rational therapy will no doubt be advanced.

Treatment of the multinodular goiter with hyperthyroidism is much more difficult than Graves' disease. The gland is resistant to radioactive iodine necessitating the administration of large doses. Thus surgical excision is considered by most as the treatment of choice. In older patients, who are poor surgical risks, a trial of propylthiouracil or tapazole followed by a dose of radioactive iodine would be appropriate.

A solitary toxic nodule is highly resistant to radioactive iodine therefore the treatment of choice is surgical resection of the nodule after the patient has been restored to a euthyroid state. Obviously cases of hyperthyroidism due to subacute thyroiditis will spontaneously resolve and those due to excessive exogenous thyroid hormone will be corrected by the appropriate lowering of the administered dose.

In conclusion, there are several forms of hyperthyroidism which can present with similar clinical manifestations; these need to be characterized so that appropriate therapy can be utilized for each. Measurements of triiodothyronine by radioimmunoassay can assist in arriving at the difficult diagnoses. In the future the response of serum TSH to thyrotropin releasing hormone will be extremely valuable in the early diagnosis of hyperthyroidism. Careful attention to older patients with cardiovascular manifestations may prove helpful in diagnosing unrecognized hyperthyroidism.

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Seminars in Chest Medicine

ARE CORTICOSTEROIDS WORTH THE RISKS IN OBSTRUCTIVE LUNG DISEASES IN ADULTS?

JAMES O. MORSE, M.D.

This continuing series of articles entitled "Seminars in Chest medicine" will attempt to keep the reader abreast of developments in the broad field of pulmonary diseases. The format used will be that of brief succinct reviews written by the editors as well as guest contributors. Areas of controversy as well as practical chest medicine will be explored. We hope that these reviews will be of value in promoting continuing education for certification examinations as well as a forum for new and controversial issues. The editors welcome comments and discussion from our readers.

ROBERT J. CLARK, M.D.
LYNN M. TAUSSIG, M.D.
WILLIAM C. WEESE, M.D.

Corticosteroid drugs have been used in the treatment of obstructive lung disorders for over 25 years. Initial enthusiasm for large divided doses over prolonged periods of time soon faded as frightful side effects appeared. Cautious physicians began to avoid using steroids whenever possible, and the fear of steroid dependence came to rival concern over iatrogenic narcotic addition. For some physicians, weaning an asthmatic from steroids seemed to be about the most important aspect of his treatment. A reluctance to reinstitute corticosteroids, even for severe attacks of asthma, may have contributed to some unnecessary mortality.

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In recent years there has been a realization that for many asthmatics, corticosteroids are not always the drug of last resort to be used only in desperate situations. Part of this change has been brought about by the recognition that the chronic administration of small doses gives few side effects, particularly if the doses are spaced out in such a way that the natural pattern of morning rise and evening fall in serum cortisol levels is not violated[1]. If doses can be stretched out to 48-hour intervals, even fewer side effects occur and near normal pituitary and adrenal function can be preserved, thereby avoiding the dangers of relative adrenocortical insufficiency during unexpected stresses[2,3]. However, acceptance of even these reduced risks could not be justified if the benefits were only questionable.

In most cases of bronchitis and practically all those of uncomplicated emphysema, objective benefits from the chronic administration of corticosteroids are neglibible.[4] In a few cases of bronchitis and in essentially all those of true asthma, the response to corticosteroids is definite and, more often than not, can be obtained with modest doses such as, 7.5 mg to 15 mg of prednisone each morning (or, preferably, 15 to 30 mg every other morning).[5]

Better understanding of the mechanisms involved in asthma helps explain why low dose corticosteroids are of value. Mast cells situated in the lungs can release mediators such as histamine and slow-reacting substance of anaphylaxis (SRS-A). Such mediators produce bronchoconstriction, mucosal edema, and excessive mucus secretion. When an antigen reacts with its specific reaginic antibody (IgE) bound to the mast cell, such a release is triggered. This has been demonstrated repeatedly *in vitro*. It appears likely that other stimuli are also available *in vivo* or that, as the disease progresses, the trigger becomes so sensitive that nonspecific stimuli are continually setting it off. Whatever the stimulus, the mast cell becomes less susceptible to being triggered if its intracellular level of cyclic 3′, 5′-adenosine monophosphate (cAMP) is maintained. Bronchodilators increase the levels of cAMP, and steroids seem to increase the cAMP, and response to the beta-adrenergic drugs.[6]

Although high and continuously elevated blood levels of corticosteroids are probably necessary to suppress the inflammatory response in bronchitis, in the chronic state of asthma relatively small amounts given at infrequent intervals may be enough to stabilize the mast cell and inhibit excessive mediator release. (Cromolyn appears to work at this level, also, although its effects are not on cAMP). In a particular patient with chronic obstructive lung disease (COPD), sputum eosinophilia appears to be the best guide as to which case merits a trial of steroids.[7] Even then a trial is hardly justified if the obstruction can be satisfactorily controlled with tolerable doses of oral and inhaled bronchodilators or, in the case of asthma, with cromolyn.

The tragedy of unnecessary steroid dependence is seen not infrequently, usually as the result of a patient with severe COPD being placed on steroids out of desperation. The drug is continued without any objective evidence of improvement in lung function, because the patient feels generally better; this sense of feeling better is, however, a common nonspecific effect of initial steroid therapy in anyone. When this initial euphoria fades, the physician changes his mind about the efficacy of this therapy, and decides to discontinue it. Of course the patient then feels worse, as he suffers the letdown of steroid withdrawal; this may trap the physician and patient into continuing a drug that was initially unnecessary. Lacking a clear end point for judging therapeutic response, the patient tends to increase the dose to dangerous levels.

Unnecessarily high maintenance doses are sometimes the result of the patient's attempt to control his disease with corticosteroids alone. Since the theophyllines are distasteful to many patients and the tremors of beta-adrenergic drugs annoying at best, there is a tendency to discontinue them or use them only if symptoms are severe. Recognizing that the only clear-cut mechanism of action by which corticosteroids have been shown to aid in the control of asthma is in increasing the capacity of the mast cell to respond to beta-adrenergic agents with a rise in cellular cAMP, it is rather foolish to discontinue or reduce the beta-adrenergic drugs as soon as a good response is obtained. If improvement allows any drug to have its dosage reduced, it should be the corticosteroids.

In other cases unnecessarily high maintenance doses are given in an effort to control completely all obstructive symptoms. It must be recognized that not even maximal doses of corticosteroids will make this possible in some patients and that the benefit of these higher doses probably does *not* outweigh the greatly increased risks of side effects. Since clearing of blood eosinophilia may indicate suppression of the release of the eosinophilic chemotactic factor of anaphylaxis, (another mediator released from the mast cells and basophils along with histamine and SRS-A,) it has been suggested that direct eosinophil counts be used to evaluate the adequacy of steroid dosage.[8] If the total eosinophil count (taken at least six hours after bronchodilator administration) is lowered to that maintained by normal individuals receiving corticosteroids (85 per cu. mm.), then larger doses would be superfluous, at least for inhibiting mediator release.

The most promising new development for the patient needing continuous steroids appears to be the inhaled corticosteroids.[9] Although dexamethasone was packaged as an aerosol many years ago, the amounts needed and its ease of absorption resulted in most of the disadvantages of oral administration plus greatly increased cost. Beclomethasone propionate or triamcinolone acetonide differs from dexamethasone in that only very small doses are needed to obtain significant topical effects. What is absorbed systemically of these drugs is probably metabolized rapidly, since pituitary-adrenal suppression is negligible. The usual starting dose for beclomethasone is only two 50mcg puffs four times a day. Up to twice this amount can occasionally be used with benefit, but less than this may be adequate in some patients.[10] Theoretically, at least, using an inhaled bronchodilator before each dose of steroids may help to distribute it to more peripheral airways. Patients with considerable obstruction may need a brief course of systemic steroids to improve ventilation to the point that the inhaled steroids can be delivered to the appropriate sites. Also, during acute exacerbations not controlled by increasing the amount of steroids inhaled, oral prednisone should be resumed for a brief course.

Mucosal atrophy has now been noted in the bronchi of patients using beclomethasone for at least a year. (Skin atrophy has been reported following prolonged use of topical corticosteroids.) Pharyngeal thrush has occasionally developed in patients using inhaled beclomethasone; gargling immediately after each dose may help avoid this complication. Another common annoyance in the patient being converted from systemic to inhaled steroids has been an exacerbation of previously suppressed non-infectious rhinitis. Some claim that exhalation through the nose following the inhalation of the steroid helps control this problem. At least it is hoped that an adapter for nasal administration will soon be marketed. Inhaled beclomethasone was marketed in the United States in the summer of 1976.

While the chronic steroid treatment of obstructive lung diseases has shifted to lower doses at greater intervals, recent studies have shown that larger doses at shorter intervals are usually needed for acute obstructive exacerbations. Serum cortisol levels between 100 and 150 mcg per ml are desirable. While these levels can be reached in normal persons with less than 100 mg of hydrocortisone intravenously, asthmatics who have been on steroids chronically may need a 4 mg/kg loading dose of hydrocortisone (or its equivalent) and 3 mg/kg in continuous intravenous infusion over each six hours.[11] There is some evidence that exogenous corticosteroids can induce the liver to metabolize administered steroids more rapidly. It is clear that phenobarbital, so commonly ingested in combination with ephedrine and theophylline, is a potent inducer.[12] In any event, it is preferable to give a generous amount of steroids initially and taper the dose as rapidly as the clinical course permits. Gradual increases in the maintenance dose are usually much less effective in getting control of an exacerbation.

In conclusion, corticosteroid treatment of obstructive lung diseases can be worth the risks involved if cases are carefully selected to include only those who are not responding satisfactorily to maximum use of other modalities and whose disease displays features usually associated with response to steroids. Risks must be minimized by careful control of the administration of the drug, both in amount and in timing. For this, careful patient education is essential.

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Grand Rounds

THE CLINICAL PICTURE OF SICKLE-C DISEASE

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CASE REPORT

T. T. a 60 year old black man was admitted to the Tucson VAH with progressive symptoms of lower extremity arthralgias and a more recent effusion in the right knee. The presence of anemia, splenomegaly and the absence of demonstrable etiology led to an electrophoretic diagnosis of Sickle-C disease. Radiologic abnormalities suggesting multiple bone infarcts were of particular interest.

Dr. Denny:

More than 8% of the nation's Black population carry the sickle cell gene and 2% for C-hemoglobin. It is estimated that 10,000 persons have S-C disease based on one per five hundred population.[1] Thus, it can be a frequently seen clinical problem in areas with a large Black population. Accurate statistics are not available, but as there are deaths and significant morbidity in patients with this abnormality; life span is probably shortened. The condition is often benign in clinical course and diagnosed during investigation for anemia, blood film abnormalities, or splenomegaly. The most common symptomatic presentations are those of bone and joint pain, or recurrent abdominal pain. Less frequent clinical features are chest or flank pain, visual disturbances, hematuria, jaundice, and priapism. Bizarre abdominal colic easily confused with ulcer, gallbladder distress, appendicitis, or pancreatitis, is common and was found in 30% of the 75 patients reviewed by River.[2] Bone and joint symptoms have been described in 50-75% and are more marked in the long bones and large joints. Splenomegaly is noted in 60-80% of the patients and is the only consistent physical finding. Less commonly noted are jaundice and retinal abnormalities. Splenic infarction may occur during exposure to high altitude or more rarely spontaneously. As in sickle cell disease there is a defect in maximal urinary concentrating ability. Both fetal and maternal mortality are increased in patients with S-C disease, but fecundity is not decreased as in S-S disease. The hematologic picture is often characteristic. In the absence of crisis or a complicating illness significant anemia is not the rule. The anemia is usually mild, although with crisis severe drops in the hematocrit can occur. The red cells are rather variable in size and the most consistent and striking finding is "target cell" formation which may exceed 80%. Because of the cell indices and morphology they are often erroneously called microcytic hypochromic as in the case presented. Careful reticulocyte counts will usually show a mild but definite elevation and some peripheral red cell polychromasia may be noted. The production index is elevated and if done the red cell survival will be about half the expected 120-day span. The sickle cell preparation is positive and hemoglobin electrophoresis will establish the diagnosis showing 50% S and 50% C hemoglobin. The fetal hemoglobin is not elevated and A^2 levels are difficult to determine because they are obscured by the C-hemoglobin migrating at the same rate as A^2.

In a Black patient with extremity or abdominal pain, splenomegaly and a positive sickle cell prep, the presence of marked target cell morphology virtually assures a diagnosis of S-C disease.[3]

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Dr. Pitt:

The radiographic findings demonstrated in the patient are characteristic of S-C hemoglobinopathy. Examination of the right knee shows early degenerative arthrosis evidenced by slight reactive sclerosis in the medial joint compartment, and narrowing in the retropatellar region. Minimal osteochondral new bone formation is present in these areas. Fluid is present in the joint and is best appreciated on the lateral view with distention of the suprapatellar bursa. The key to the diagnosis of S-C disease centers on the multiple areas of increased bone density in the distal femur and proximal tibia. Complete examination of the right femur discloses an extensive medullary infarct with well defined sclerotic margins and serpiginous linear areas of increased density. Separation from the endosteal surface in the mid portion of the shaft is noted. Focal thickening of the peripheral surface of the shaft indicating previous periosteal reaction is noted. (fig. 1)

The major radiographic findings in S-C disease reflect ischemia and are found in the osseous system.[4] These consist of bone infarcts in various stages and modeling deformities.[5] Factors which influence the presentation include the site(s) of involvement, the age of the patient at the time of ischemic insult, the length of time the lesion has been present, and the repair reactions of the host.[6,7] Both the peripheral and axial skeleton may be affected. Abnormalities in the peripheral skeleton are characteristically multiple, symmetric and frequently extensive. An increase in the number of lesions is noted with advancing age. In the adult, the medulla and endosteal aspects of the cortex are typically involved; the peripheal cortex is less affected due to the protective collateral circulation of the periosteal vessels.[6] This periosteal collateral circulation is less developed in the young and accounts for the more extensive cortical involvement in this age group.

Involvement of subarticular bone most commonthly occurs in the hips ("aseptic necrosis") and is often bilateral.[5] Collapse of this devitalized bone with subsequent loss of support for the overlying articular cartilage may produce secondary degenerative arthroses.[7]

Repair mechanisms explain the varied presentations of bone infarcts. Revascularization begins peripherally and tends to separate distinctly the lesion from the adjacent normal bone. This is followed by reossification and resorption of dead bone. The reossifying process produces the characteristic areas of increased density identified in bone infarcts;[7] bone deprived of its blood supply is nonviable and tends to maintain its pre-infarction density.

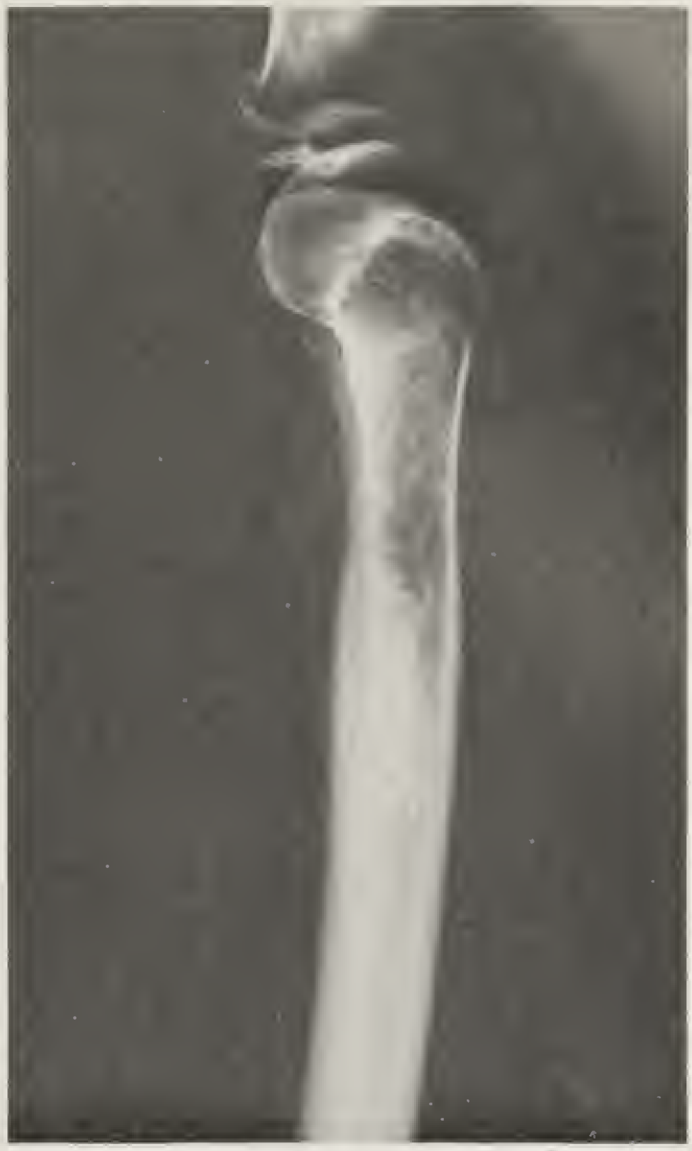

Figure 1

A well marginated medullary infarct is present in the distal third of the right femur demonstrating typical serpiginous calcifications. Widening of the medullary canal at this level is seen with focal periostal new bone formation posteriorly. Additional infarcts are seen both proximal and distal to the main abnormality in various stages of repair.

Step-like depressions of the mid portions of the superior end plates of the spine are pathognomonic changes occurring in sickle hemoglobinopathies. These are most commonly seen in the dorsal-lumbar regions. Reynolds has proposed that these changes reflect chronic ischemic underdevelopment; the more peripheral portions of the end plates receive collateral periosteal blood supply and develop in a more normal fashion.[8] Diffuse osteoporotic changes represented by a decrease in the number of trabeculations and biconcave depressions of the end plates can be seen in S-S hemoglobinopathy and reflect the magnitude of the anemia.[4] These latter changes are not generally seen in S-C disease in the United States but have been noted in countries where additional factors such as malnutrition may also be present.

Recognition and correlation of the radiographic findings in S-C disease are important since the disease may be unrecognized due to the mild clinical course.[5] Of interest is the frequent absence of previous symptoms in areas of extensive infarction and the observation that the extent of bony involvement often shows little correlation with the clinical severity of the anemia.[9]

Dr. Denny:

Pathophysiology Sickle-C Disease:

In 1946 the observation that deoxygenated sickle cells were birefringent under the microscope and lost this property with reversal of sickling on aeration was brought to the attention of the physicist, Dr. Linus Pauling. This led him to the evidence that the hemoglobin of sickle cells had different physicochemical characteristics from normal, and the term "a molecular disease" was coined.[10] At about the same time, Neel published family genetic studies proving the disease to be a homozygous state, and that the heterozygous condition was relatively benign.[11] In 1957 Ingram's "fingerprint" method of aminoacid analysis proved a single aminoacid substitution at the sixth position from the N-terminal of the sickle cell beta chain differentiated this abnormal from normal hemoglobin. This single molecular alteration permits deoxygenation to in some way cause stacking of the hemoglobin molecules in the helix of high pitch and the sickle crystal formed. Gelling, increased viscosity, and sickle crystalization are quantitively related to the concentration level of deoxygenated sickle hemoglobin. In vitro sickling does not occur until this concentration is above 10 grams per 100 ml. The relative importance of viscosity vs. mechanical effect of the sickled cells in producing microvascular occlusion remains unclear, but both must be important in the pathology observed.[12]

In the normal person hemoglobin will crystalize when its concentration is not reached during normal red cell aging. C-hemoglobin, on the other hand, spontaneously crystalizes at the concentration of hemoglobin which can be found physiologically in senescent circulating red cells. With this condensation the cell becomes rigid and is subject to splenic or RE cell entrapment and destruction. The condensation of hemoglobin within the cell produces a prominent tendency for central deposition of hemoglobin into a "target" morphology. One can readily demonstrate in vitro intracellular crystal formation by suspending red cells in hypertonic saline, or in vivo after splenectomy.[13] Individuals homozygous for hemoglobin-C thus have a mild hemolytic process associated with splenomegaly. Heterozygotes of C and normal hemoglobin display marked targeting, but no hemolysis is found. When C-hemoglobin is additive to another abnormal hemoglobin (i.e. Sickle), both sickling and target cell formation are prominent. In addition to shortened red cell survival, S-C disease is characterized by the same tendency to microvascular occlusion found in sickle cell disease of classic description. As the anemia is more mild in S-C than S-S disease, the concentration of sickle hemoglobin is higher. In vivo and vitro spontaneous sickling is related in part to the absolute concentration of sickle hemoglobin, thus S-C patients have as frequent or more frequent episodes of microvascular occlusion leading to bone infarcts, painful crises, retinal and renal lesions. As with sickle cell disease, patients with S-C disease have a renal concentrating defect which cannot be reversed in adults despite exchange transfusion of normal for abnormal red cells.

Management:

The crises of sickle cell or sickle-C disease come suddenly and the patient can neither prevent nor abort them. Infection must be ruled out whenever symptoms of crisis appear and treated vigorously when present. Symptomatic measures of oxygenation, correction of acidosis or dehydration as well as relief of pain are important. Partial exchange transfusion is an important therapeutic tool for it seems to interrupt the vicious cycle of sickling. Childbearing is an acceptable risk in sickle-C disease but oral contraceptives are probably safer than an unwanted pregnancy. No drug is now recognized as an effective agent for the prevention of termination of painful crises. Sodium cyanate is deserving of continued clinical investigation. Long-term exchange transfusion treatment needs careful evaluation, but the risks and expenses are major. Public education measures and sympathetic genetic counseling of individuals at risk must be central to any preventive health measures.[14]

Exciting, but as yet highly experimental, are methods of altering gene function, transplantation of normal marrow, or attempts to induce resumption of fetal hemoglobin formation to counterbalance the effects of the sickle hemoglobin.

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Department of Health Services

COMMUNICABLE DISEASE LEGISLATION

SUZANNE DANDOY, M.D.
DIRECTOR

Among major health-related legislation passed by the last session of the 32nd Arizona Legislature are two relating to communicable disease that merit more than a passing glance. They are House Bill 2026, mandatory immunization, and Senate Bill 1022, tuberculosis examinations.

Senate Bill 1022, which was sponsored by State Senator Jack J. Taylor, R-Masa, removes the requirement that school employees submit to periodic examinations for tuberculosis. It also includes the exception, requested by this Department, that a school board may require an employee displaying symptoms of pulmonary disease to submit to such tests or examinations as a licensed physician deems appropriate. The bill became effective May 12, 1976.

Introduction of the bill followed closely the September 1975 elimination of such testing as a condition of employment in the food industry, health institutions and schools of Maricopa County by that government's Board of Health.

Public health agencies concerned with detection and prevention of tuberculosis have, in recent years, begun to question the cost effectiveness and productivity of such routine testing as a screening mechanism. Consistently, the case-finding productivity is very small. For instance, not one active case of tuberculosis was found in the 2,220 tests administered to teachers and school employees by the Maricopa County Department of Health Services during 1975. Considering the manpower, time and equipment necessary for mass testing, these results simply did not justify the cost. The Department of Health Services Tuberculosis Control section has for some time recommended discontinuance of annual x-ray testing of groups not considered to be at high risk of contracting the disease. House Bill 2026 requires the immunization of school children prior to the first enrollment in a public school. The bill also transfers primary responsibility for the school immunization program from the school districts to county health departments. The law is effective beginning with the 1977-78 school year.

School entry is presently the only point at which direct contact with all children is possible and where the location of individuals in need of immunizations can readily be accomplished. It is also the place where a large captive group exists, thereby facilitating the effective elevation of population immunity.

Since 1972, the responsibility for providing immunization services has been with the elementary school districts. Vaccines are provided by State funds through this Department and administered under State guidelines. Personnel administering the vacines are school-assigned or county health department nurses, or a cooperative combination of the two, all under physician supervision. Participation is strictly voluntary.

The record shows that the present program has had considerable success. Consider: only one case of paralytic polio has been diagnosed in Arizona since 1972, although 20-25 percent of the children entering school have received less than three doses of vaccine; diphtheria is not a problem among Arizona children, despite the fact that 15 to 20 percent of children entering school have received less than three doses of toxoid; measles morbidity rates during 1975 were 3.8 per 100,000 in this state as compared to a national average of 11.3 per 100,000; and there were no cases of rubella syndrome of the newborn reported in Arizona during 1975.

Overall, the immunization levels for preschool children in the state are approximately 70 percent, a commendable figure.

But, with a well-enforced mandatory program, it is anticipated that immunization levels by the end of the first year of school should be between 85 and 95 percent, well above the levels that appear necessary to achieve so-called "herd immunity." (Although the exact percentage at which the "herd immunity phenomena" becomes operative is not known, experience indicates that epidemics cease to occur in populations when the immunity level reaches 75-80%.)

Under the new legislation, this Department is charged with developing and adopting rules and regulations for administration of the program. Accordingly, we are presently drafting these guidelines to prescribe initial immunizations, recommend optimum ages for inoculation, and develop forms and procedures for a uniform system of administering, collecting, recording, reporting, and case follow-up. A final draft of these rules and regulations will be ready in October.

Physicians and other interested persons are invited to submit suggestions and comments relative to development of these rules and regulations, both prior to and during the public hearing. Please direct your comments to Jon Counts, Dr. P.H., chief of the Bureau of Disease Control and Laboratory Services, Arizona Department of Health Services, 1740 West Adams, Phoenix, Arizona 85007.

THE BADGE OF MERIT

No doctor's office can achieve esteem
Without diplomas hung on every wall.
The greatest number that one can install
Gilt frames their varied qualities redeem,
For no one ever reads the dreary theme.
Why are they hung? Aesthetic worth is small
And doctors never advertise at all.
Perhaps they mean achievement of a dream.

If this be so then let no earthly powers
Mock him whose name these framed diplomas
bear.
Still, sometimes when my baser passions rule,
I think I'd like to steal in after hours
And place another framed diploma there
To signify success in Sunday School.

Stanley Crosbie, M.D.

Radiology Case of the Month

CASE NO. 14

GLENN C. COOK, M.D.
GERALD D. POND, M.D.

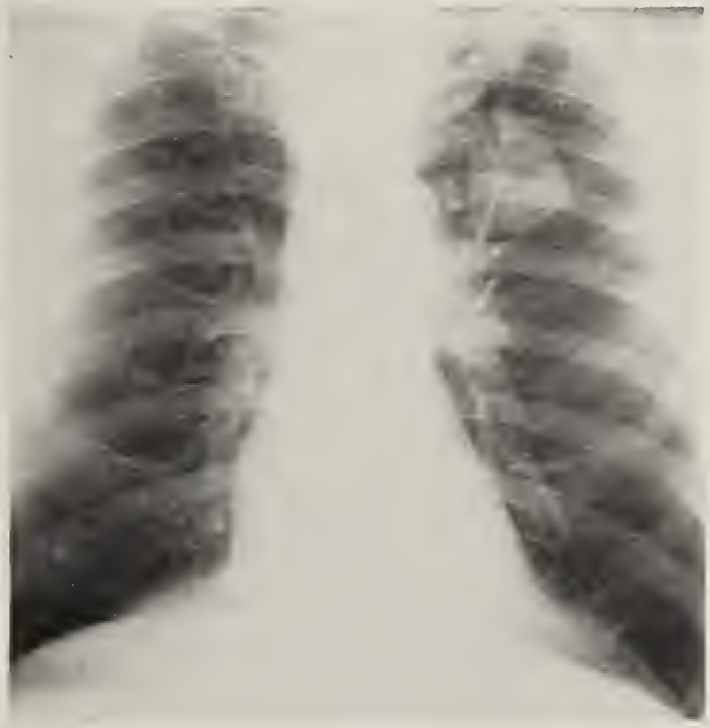

Figure 1
Chest

This is a 55 year old man with known carcinoma of the lung (Figure 1). He presented with a two week history of back pain.

Physical examination revealed a thoracic kyphosis and tenderness to palpation of the mid-thoracic region. The patient was afebrile.

Tomograms of the thoracic spine were obtained (Figures 2 and 3). What are the findings, and what is the most likely radiographic diagnosis?

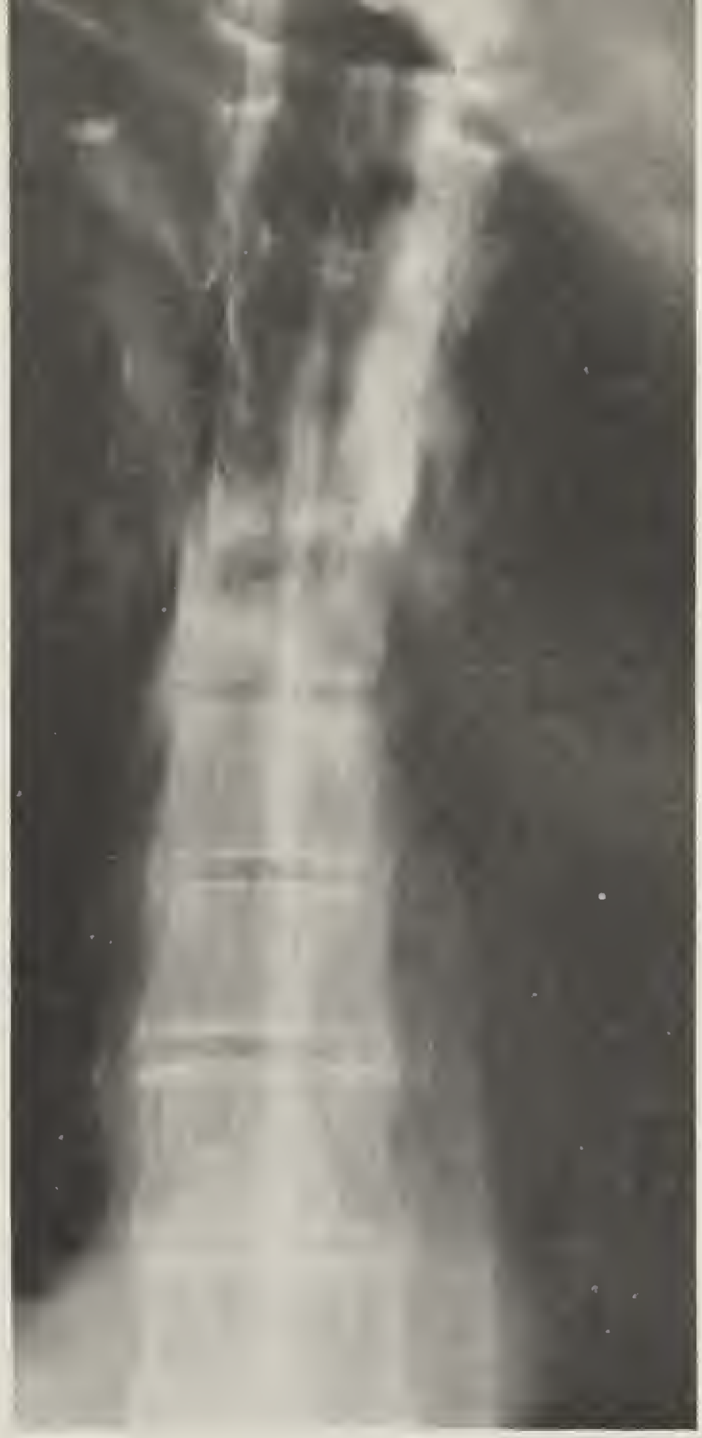

Figure 2
AP T-Spine

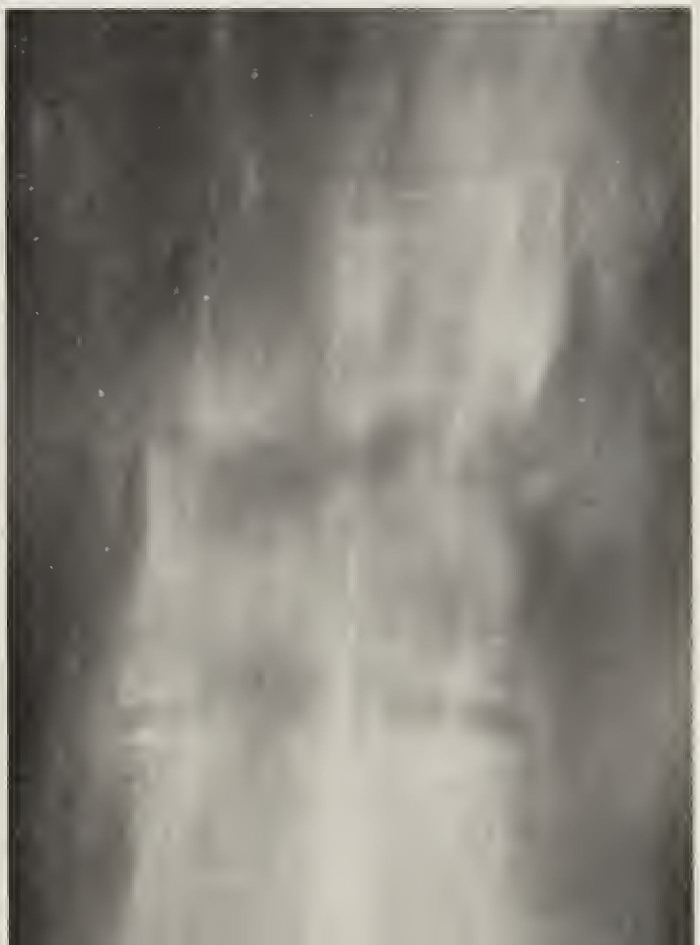

Figure 3
T-Spine Blowup

INFECTIOUS SPONDYLITIS
[Actinomyces israelii]

The chest reveals a left upper lobe mass consistent with carcinoma. The tomograms demonstrate destruction of the vertebral body and end-plates at T3-4 and near complete obliteration of the intervertebral disc. The borders of the destructive process are ill-defined.

The working diagnosis was metastatic involvement of the spine. However, metastatic disease only rarely crosses a disc space even though it commonly involves the spine. The relative avascularity of the cartilage provides a barrier to invasion. Infectious spondylitis, on the other hand, tends to begin at the vertebral end-plate and destroys the disc either enzymatically (by lysosomal enzymes) or by toxins elaborated by the organism. Therefore, infection is the more likely radiographic diagnosis.

Biopsy confirmed an infectious process, and *Actinomyces israelii* was isolated. Follow-up studies 6 months later after antibiotic therapy show evidence of bone repair consistent with a healing inflammatory process (Figure 4).

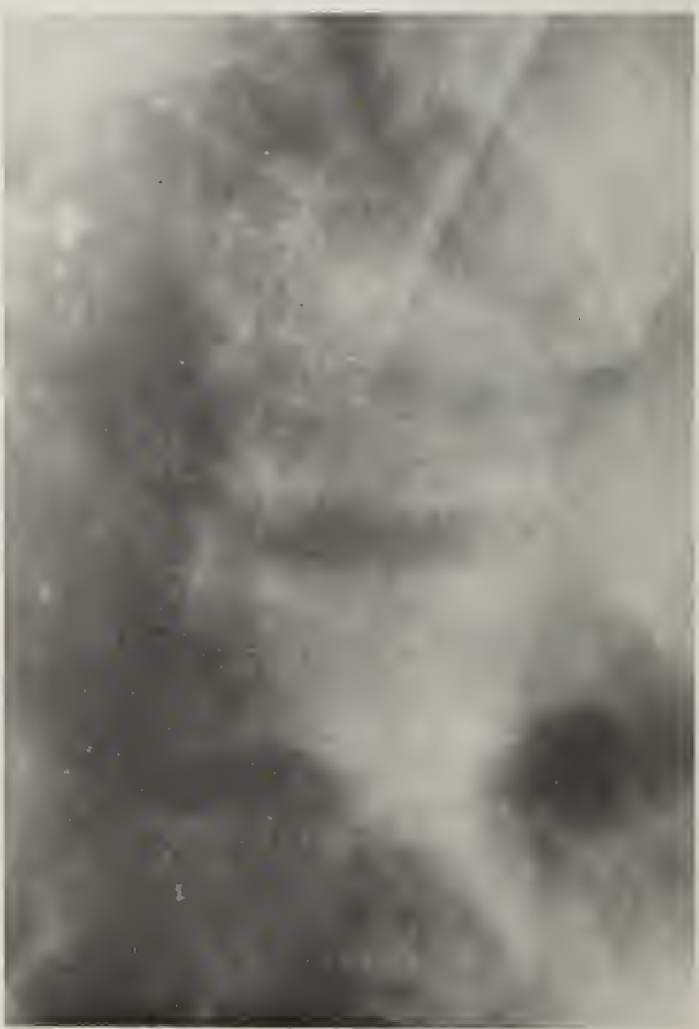

Figure 4
LAT T-Spine

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Editorial

THE EXPERT

WILLIAM B. McGRATH, M.D.

For the development or repair or appropriate use of any kind of equipment there is somewhere an expert. Similarly there is a mentor for whatever activity one might undertake, from sports to crafts to the most highly skilled professions.

Indispensable to the expert are any or all of the following traits.

1. In his field he has learning and skill. This, if you will recall, is the definition of wisdom, the intelligent application of knowledge. Implied are his familiarity with the history of his subject and broad basic information about anything which relates to it.

2. He is highly specialized. As his upward trail narrows he can touch the sides of his subject almost intimately. To be an expert is to climb to the top of a particular peak in the mountain range of more generalized competencies. (The discoverer of Librium and Valium, Leo Sternbach, was not just a chemist but the one who best understood the chemistry of those compounds.) Surely not excluded from consideration is the general practicioner of medicine. He is supposed to be a specialist in treating the whole person.

3. The expert has dignity. One cannot be influential and at the same time joking and familiar. Even the saloon-keeper intuitively knows better than to serve secular "communion" on the customers' side of the bar. If the expert truly understands his role and whenever he is functioning in it, he must be taken seriously.

4. He puts a very high value on virtue. There are sound and practical reasons for this. Only the virtuous person has some command of his own destiny. The expert has to have manifest control of himself and of his own drives and appetites (temper, for example, or sexual or drinking habits.) If he is not respected he cannot exercise authority or expect to inculcate those disciplines which his clients or charges will require.

5. He is articulate. Individuals of acknowledged genius almost always prove to be uncommonly literate. Only by teaching or writing does one keep his own knowledge organized and his viewpoint in focus. Expertise in solitude, unchecked, can sour or go astray or become fanatic.

6. He is not a know-it-all. Though he has warranted confidence in his own specialty, he claims no monopoly. There are boundaries of his chosen subject and he is never dogmatic beyond them.

7. He is not offensive. He never finds it constructive to put his client or patient or customer on the defensive. He would default his role and defeat his purpose by whining criticism or rebuke. Only in exceptional circumstances might he volunteer or intrude without invitation.

8. He is not arrogant. His relationship to his client is not as master to pet or parent to child or superior to inferior. The expert sets himself not above but somewhat aside from his clients. Perhaps they, too, are experts but in other fields. Freud compromised this subtle distinction in the ritualistic arrangement of the couch and the analyst.

Now the expert fixes some piece of equipment or shows a person how to do something. When a trusted mechanic reports that your car is functioning efficiently you are relieved of doubt and worry. The trainer says that your horse is sound and the riding instructor signals that you are ready to mount. There is a lightening of your heart because the professor certifies that you are qualified for graduate school.

The opinion of an expert does not afford just *relief*. It is also a *release*. It is an authorization to go ahead and use the equipment or to practise and enjoy the skill one has learned. Only the expert has this authority.

For those who profess expertise in human affairs there is another and really exalted exchange. From such an expert a person seems to asking for evaluation or advice. Far more profound is the client's need of *acceptance*.

However we may appear to others we are all oppressed by self-doubt and the dread of rejection. No one can simply and smugly forgive and accept himself or have any firm concept of his own worth.

So we grant authority to a somewhat impersonal agent who is qualified to have an expert opinion. When we feel accepted by the expert it becomes a little easier to accept ourselves. We are paroled. We have received permission to put our own capabilities to use.

From: 207 E. Monterey Way, Phoenix, AZ 85012

9. He is objective, impersonal—as one pictures the priest in the semi-darkness of the confessional. The expert realizes that his own personality is really not involved and is unimportant. There is no call for him as a person to become righteous or overbearing.

10 He is tough, though not belligerent. Any successful apprenticeship will have entailed extraordinary tenacity and endurance, and some competitiveness. The expert also has to be sure of his own integrity, sure that there is a line beyond which he will not go and cannot be pushed or seduced; and he has to convey (silently) to others the fact that he can never be bribed or intimidated.

11. He has a kind of simplicity. His motives are not complicated. He has colleagues instead of competitors. He is past having to prove anything or boast or posture. He uses his skill "because it is there" and because it is needed.

The expert is not a messiah, of course. Yet when we are considering the client-expert relationship we come close to the very heart of religion: If one can accept a forgiving God one can believe one's own forgiveness; if one feels acceptable to God, one can accept one's self.

On a very finite or miniature scale of any exchange between a client and an expert. Everywhere such transactions are occurring. In the sublimest meaning of the term there is a wee bit of religion in very instance of helpful acceptance. Awareness of this might well be the foundation of the philosophy of a professional man.

PSYCHO-FORENSICS
ON THE MENTAL CONDITION AT THE TIME OF THE CRIME

An attorney or the court frequently asks a physician, and particularly a psychiatrist, to give the mental condition of a person on or about the time of a legally significant event. In his report or in his testimony, this physician must prove his "findings", or his diagnoses, by satisfying the legal standards of proof, so that in a civil case his diagnosis must be derived from the preponderance of evidence, and in a criminal case must be beyond reasonable doubt—using essentially the same reasoning that he would follow in making a medical or a surgical diagnosis. Since it is most unusual for the physician to have examined the subject at the moment of the event, for example at the signing of a will or committing the crime, and since examinations immediately before or after such an event are rarely done, a certain amount of medical judgement is required for him to give an answer to the legal question in a manner that is accurate, or even true.

Probable Mental Condition

In order to precisely diagnose the mental state of an individual at some moment in the past, we must plot the progress of that individual's development, after eliciting the history of the physiological, psychological and social forces that have governed him. Then as medical experts who are familiar both with the natural history of disease and the natural history of normal mental functioning, we simply move backward or forward along the abscissa of that plot to the moment of the legally significant event. The expert may then follow certain rules of clinical judgement[1] or decision analysis[2,3] and may utilize the terminology that is available[4] in the Diagnostic and Statistical Manual of Mental Disorders, published by the American Psychiatric Association. This Manual gives diagnostic criteria for all or most mental disorders, and then toward the end, refers to normalcy as follows:

> 318 No Mental Disorder
> This term is used when, following psychiatric examination, none of the previous disorders is found. It is not be be used for patients whose disorders are in remission.

The Manual does not mention the criteria for remission, but does deal with this topic in discussing the acute schizophrenic episode—a subject of some forensic interest— "In many cases the patient recovers within weeks, but sometimes his disorganization becomes progressive. More frequently remission is followed by recurrence."

Thus the "probable mental condition" of an individual at a specific time, be it normal or abnormal, can be diagnosed by using ordinary logic and medically acceptable terminology. Yet while the responsibility for the diagnosis of No Mental Disorder, for example, is in the hands of the physician, who knows that normal mental functioning was found in only 20% of a large group of well studied individuals[5] and that normalcy itself may be an ideal fiction[6], in our legal system it is the responsibility of the jury or the judge to find the facts of this legal matter. Bluntly put, the jury or the judge make the diagnostic determination, whether it be one of health or one of disease.

The Improbable State

Viewing the other side of this question, we see three major factors that can each invalidate the retrospective diagnosis of an individual's mental condition at the time when he was involved in an act which now brings him before the bar—the frailties of human memory, the expectable variations in cerebral functioning, and the tendency to place self-preservation above the truth.

The difference between forgetting and amnesia has been assayed in an experimental study on alcoholics who suffered from what they described as blackouts. Goodwin[7] suggested that amnesia is a loss of memory for events which one would reasonably expect an individual to remember, like punching a man in the nose at a bar while drinking, but that forgetting is a loss of memory for items that may reasonably be lost, like "Just when *is* Aunt Jennifer's birthday?". Working with hysterics, Sigmund Freud observed that "Neurotics suffer from reminiscences", and learned how the conscious memory of traumatic events is repressed in all of us, as the trauma's traces are deposited in the unconscious. While older people show a diminution in the acuity of memory, education gives some definite protection against this diminution[8] and compulsiveness, which is in part a self-educative process, also protects against memory decay. Certainly an event that has currently come before the court would be a valid candidate for the processes of amnesia, blackout, repression, or the vagaries of a senile testator's memory.

Cerebral function, and simultaneously memory function, varies in the normal through the stages of sleep which follow a 90 minute cycle, to wakefulness. In persons with a generalised seizure disorder, convulsions are a relatively rare event, and status epilepticus, with unremittingly continued generalized seizures, often with hyperthermia and a fatal outcome, is extremely rare. Great changes take place in the mental functioning in these, and many other conditions of health or of disease.

As for the battle between self-preservation and the truth, litigants and juries alike know that the cup which is half full is also half empty, and have long been accustomed to seeing the big strawberries on the top of the basket. At the other extreme, lying, malingering and simulation of health are too often the warp upon which the woof of medico-legal histories are woven.

Thus when the court asks the doctor, "What was the mental condition of this person on that date and time?", the doctor may quite correctly reply, "I don't know." **WFG**

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Topics Of Current Medical Interest

TALMADGE MEDICARE-MEDICAID REFORMS AIMED AT CUTTING RISING FEDERAL COSTS

Attorney General Edward H. Levi was quoted recently as saying, "We must protect ourselves against a kind of bombardment of demand for instant solutions that keeps us from working things out."

Mr. Levi was talking generally. But a mile from his office, Senator Herman E. Talmadge is pressing for just that kind of instant solution to the rising costs of Medicare and Medicaid.

A bill sponsored by him could have an adverse effect on the delivery and quality of care under those programs, without alleviating the costs.

Under the Talmadge bill, Medicaid fees paid physicians for outpatient care would be not less than 80 per cent of "reasonable" charges under Medicare. This may sound attractive, Since Medicaid fees in some states presently are as little as 50 per cent of the Medicare figure. But are Medicare charges fair? And wouldn't the 80 per cent figure make doctors continue to bear the onus for Medicaid cost overruns?

Certainly there should be reasonable efforts to arrest the costs of Medicare/Medicaid. But many of the provisions in the Talmadge measure are a gauntlet "that keeps us from working things out" (to borrow again the words of the Attorney General).

Also under the bill, there would be two assistant secretaries of health handling the programs—including a new one to head a Health Care Financing Administration. This would include an Inspector General with authority to review and audit all health programs covered by the Social Security Act.

Such extra bureaucracy and red tape would in themselves be an expense, without adding appreciably to powers already invested in HEW.

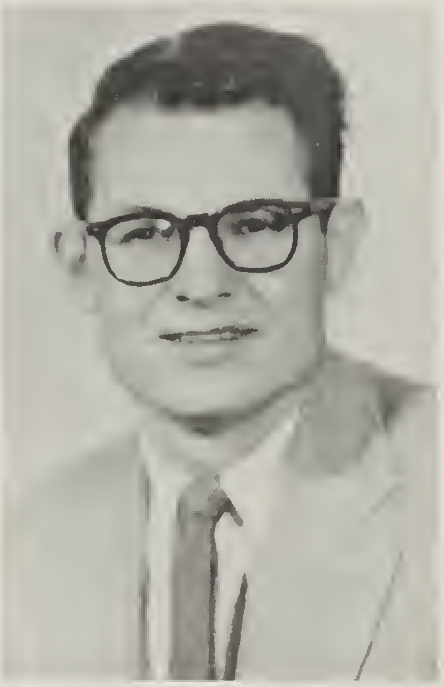
Hospitals all across the country would be classified and categorized, under the Talmadge bill. Reimbursement patterns for Medicare/Medicaid cases would be geared to the average costs of each class of facility—despite the fact that costs largely reflect the caliber of services provided.

Obviously there would be a tendency for hospitals to peg their costs to the average, thus lowering quality.

Besides, hospitals in many states are undergoing fiscal hardships because of present payment levels under Medicare/Medicaid. The Talmadge bill could squeeze some of them to death.

The AMA House of Delegates and Board of Trustees have issued a strong critique of the bill, and Association representatives will testify on it at hearings this summer.

AM In Memoriam



TELFORD K. CHAPMAN, M.D.
1931—1976

Telford King Chapman was born 25 August 1931, the fourth and last child of Ernest Collins Chapman and Mary Jenette Adair, in Bluewater, Valencia County, New Mexico. He was left fatherless in 1938 at the age of seven, when a tragic accident took the life of his father.

He grew up in Bluewater and attended grade school there. In 1945 he began High School at Grants, New Mexico. Two years later his family moved to Mesa, Arizona where he graduated from Mesa High School the 27th of May, 1949.

Following High School he enrolled in what was then known as Arizona State College in Tempe, Arizona for a year. He was a member of the ROTC and took pride in his training. This added to his desire to serve his country, and in June 1951, after the Korean conflict broke out, he enlisted in the U. S. Navy, probably with the encouragement of his elder brother, where he served for 3 1/2 years.

He was stationed at Treasure Island and his tour of duty took him to all parts of the Pacific as an Electrical Technician on board the destroyer, U. S. S. Perkins. His interest in medicine was born aboard ship as he watched his buddies die following the shelling of his ship off the coast of North Korea.

His tour of duty was pleasantly interrupted in February, 1953, when he was granted a month's furlough to marry Carol Christensen in the Arizona Temple.

He was honorably discharged from the Navy in 1954 and at this time he enrolled at the University of Utah in Salt Lake City. In 1958, he graduated from this University with a Bachelor of Arts degree in Physics and Math. He was accepted into Marquette University School of Medicine in Milwaukee, Wisconsin and graduated in 1962 as an M. D. He immediately returned to Arizona to do his internship at St. Joseph's Hospital in Phoenix. During this time he also served as a part-time Doctor for the copper mine employees in the Globe-Miami area as well as an Emergency Room Physician in the Baptist hospital in Phoenix, and operated the Pinal County Clinic at Casa Grande along with his good friend, Dr. William Russell.

After completing his residency in 1966, he moved to Mesa with his family and set up practice across from the Lutheran Hospital. Later the demands of his practice required the move to his present location on West 5th Place.

Telford is the father of two girls, Susan - age 20 and Dana - age 13, and two boys, Telford and Mark, both aged 9.

He passed away June 26, 1976 of Cerebral Thrombosis.

Friends are setting up a medical memorial fund in his name to assist a worthy student in the medical profession.

He belonged to the Maricopa County Medical Society, the Arizona State Medical Society and the National Medical Society. He also worked with the Maricopa County TB Society.

Letters to Editor

John W. Kennedy, M. D.,Editor
Arizona Medicine
Dear Dr. Kennedy:

I was dismayed to learn (in the June, 1976 issue) that *Arizona Medicine* is in "guarded condition"! Although I am not a member of the Arizona medical community, I am a member of the large number of persons engaged in disseminating information — a reference librarian at the University of Arizona Main Library.

Arizona Medicine is indexed in one of our basic indexes, *Index Medicus,* copies of which are located in the Science Library and the Medical Library at the University Medical College. Thus, there is ready access by subject to the articles. In addition, the University Library has, for many years, been compiling a unique tool called the *Arizona Index*. I am the compiler at the present time. On a regular basis I index articles about Arizona which appear in approximately sixty journals, within a wide variety of subject fields. This Index is in card form and is duplicated for ten other libraries within the State, five of which are in the Greater Phoenix Area. From *Arizona Medicine* I index the excellent historical articles, information about Arizona physicians such as from "In Memorium," and items about special research and educational programs.

The journal is so excellent I should certainly be sorry to see it die, or to deteriorate to a newsletter-type publication. Perhaps it could be published less frequently — bimonthly, or even quarterly? I hope so!
Sincerely yours,
(Miss) Virginia E. Rice,
Social Sciences Reference Librarian

George D. Comerci, M. D.
Associate Dean, Continuing Medical Education and Outreach
University of Arizona
Tucson, Arizona 85724
Dear George:

Thank you for a copy of your note of July 9, 1976 to Dr. John Kennedy, editor of *Arizona Medicine*. I think that the points you made are well taken. I don't know how much of an impact this may have, but I would certainly agree with your feelings. With relation to the fact that the authors could pay ten to fifteen dollars per published page, I would add that if this is accepted, it should be a negotiable item since some individuals, such as fellows or interns and residents, might be waived from this assessment. Also it would then be useful to listen to what the authors of the various papers have to say since they worked actively contributing to the overall attractiveness of the magazine, not just by means of their articles, but financially as well.

Sincerely yours,
James J. Corrigan, Jr., M. D.
Professor of Pediatrics
Chief, Section of Pediatric Hematology-Oncology

John W. Kennedy, M. D., Editor
Arizona Medicine
Dear Dry Gulch:

Despite what Flip Wilson said "what you see is what you gets," there are intangible efforts that should come from "Arizona Medicine." These could include a little gas (reportable) with the dew. For the consideration of the Editorial Board:

1. Scientific articles will be considered only as they relate to medical problems of Arizona, and that realtionship will be concisely demonstrated, i. e., articles on cocci, effects of state health programs on health (premature transport of distressed newborns resulting in failure of usual cardio-reps. mechanisims to adapt to a usual ambient environment). The effectiveness of health education programs in changing determined behavior of the population. Cost effectiveness of various enviromental programs — despite demonstrated of optimal,i. e., low. flourides for up to age 16-20, the added benifits to post menopausal females and males from high flouride content of drinking water and innumerable enviromental health programs. Can they demonstrate cost effectiveness.
2. Analytical articles concerning local, state and national legislation. How fast can the editor react to current interest topics. Would the board delegate final approval to the editor?
3. One general practioner scientfic article per month, consisting of a review of the literature, present status as it relates to Arizona and submittedly an Arizona Medical Student, in or out of U. S.
4. One article on the history of medicine, preferably in Arizona, but world wide are acceptable and compete in a separate category for alternate months or quarterly as necessary. History is recognized as an event that occured in 1976 or 1876, or 1776. If earlier it should relate to medicine in Arizona.
5. Anectodotal tales of our 50 year members and an attempt to get anectdotes from Arizona pioneers as to how they remember medicine in pioneer days.

No comment on the subjects you also mentioned in "Arizona Medicine"—"To Be or Not To Be."

What is the purpose of a state medical journal — must be localized. The above localized, but I recognize the other items you suggest are of great locat (State) interest. If you need help in the above items I suggested I would be happy to be a gad-fly or a honey bee.

Sincerely
Louis C. Kossuth, M. D.

ArMA Reports

EXECUTIVE COMMITTEE

The Executive Committee meeting of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, AZ on Friday, June 11, 1976 convened at 6:59 p.m., Edward Sattenspiel, M.D., President and Chairman, presiding.

MINUTES

The minutes of the meeting held April 27, 1976 were approved as distributed.

LAB AND X—RAY INTERPRETATIONS

The committee reviewed Lawrence J. Shapiro, M. D.'s letter of 5/18/76 written in response to Rudolf Kirschner, M. D.'s letter suggesting that perhaps some laboratory and x-ray reports may not need consultation reports.

It was determined to forward Dr. Shapiro's letter to Dr. Kirschner and to the hospital association for their information.

BOMEX APPOINTMENTS

It was determined not to submit additional nominations to the Governor at this time.

EXTENDED ROLE OF THE NURSE

Dr. Fulginiti's letter of 5/21/76 regarding the recent moratorium on certifying nurses in the extended role was discussed.

IT WAS MOVED AND CARRIED TO REFER THIS MATTER TO THE SECTION ON ALLIED HEALTH PERSONNEL OF THE PROFESSIONAL COMMITTEE FOR REVIEW AND RECOMMENDATION.

ROBERT L. ANDERTON, M. D. LETTER

Dr. Anderton's letter regarding the building fund and malpractice insurance was reviewed.

It was determined to write Dr. Anderton regarding all the activities in which the Association is engaged to attract physicians to Arizona.

PUBLIC EDUCATION ABOUT HEALTH

The announcement of Ms. Sabina Brown's recent appointment as assistant for public education at the Arizona Medical Center was noted.

FINANCING LEGISLATORS

Dr. John Wadleigh's recommendation that the Association sponsor a fund to assist physicians who want to run for the state legislature was discussed. It was pointed out that such a fund sponsored by the Association is probably illegal.

It was determined to refer this matter to ArMPAC and also urge Dr. Wadleigh to head up a private group not under the sponsorship of the Association.

UNNECESSARY AIR TRAVEL EXPENSE

Resolution 27-76 on the subject matter was discussed. It was agreed that the Executive Committee would recommend to the Board of Directors that the policy of the Association be to reimburse those traveling by air for the Association at coach fare rates.

INSTALLMENT PAYMENT OF DUES

It was decided to recommend the following payment schedule to the Board.

		Due	Delinquent
First payment	$125	January 1st	February 15th
Second payment	$125	April 1st	June 15th

MARSH AND MCLENNAN

Mr. Robinson reviewed Marsh & McLennan's offer to fund research into the causes of malpractice by providing up to $5,000.00 per quarter.

IT WAS MOVED AND CARRIED TO REFER THIS OFFER TO THE MALPRACTICE INSURANCE CRISIS COMMITTEE.

MALPRACTICE ASSESSMENT

Mr. Robinson reported that the following members who had been suspended for non-payment of the 1975 special assessment have subsequently paid the assessment and have been reinstated as members in good standing.

MARICOPA
Wilbur C. Bayham, M. D.
Paul S. Causey, M. D.
Robert B. Cloar, M. D.
Milton M. Connell, M. D.
Michael M. Daftarian, M. D.
George W. Gannon, M. D.
Richard A. Melde, M. D.
William O. Minturn, M. D.

PIMA
Raymond F. Bock, M. D.

PINAL
James H. Boyd, M. D.

YUMA
Robert G. Delph, M. D.
Maurice S. Goldman, M. D.

FLUORIDATION

Dr. Kahle reviewed the letter from Gill Read, D.D.S. of Flagstaff requesting that we write to the Editor of the Arizona Daily Sun congratulating the citizens of Flagstaff on their new water fluoridation program.

IT WAS MOVED AND CARRIED TO WRITE SUCH A LETTER SETTING FORTH THE POSITION OF THE ASSOCIATION ON FLUORIDATION AS ADOPTED 9/21/74.

BOARD OF DIRECTORS AGENDA

The Committee reviewed the agenda of Directors meeting scheduled for 6/12/76 and developed several recommendations.

Meeting adjourned 8:42 p. m.
William E. Crisp, M. D.
Secretary

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, AZ. on Saturday, June 12, 1976, a quorum being present, convened at 10:08 a. m., Edward Sttenspiel, M. D., president and chairman, presiding.

WELCOME

Dr. Sattenspiel welcomed the several county society presidents and other guests.

MINUTES

The minutes of the meetings held April 27, 1976 and May 1, 1976 were approved as distributed.

PHOENIX POWER SQUADRON

Donald F. Schaller, M. D. and Commander William Hurd of the Phoenix Power Squadron presented a painting "Sea and Rocks" by Marge Young to the Association in an expression of appreciation for the use of the building for their meetings.

ArMPAC

Dr. Langston reported that membership is about one half that of last year's and consequently funds for candidates are low. He reported that at their last meeting funds had been approved for numerous state candidates of both political parties. He urged all members of the ArMA board to become sustaining members of ArMPAC.

BOARD OF DIRECTORS

Status Report on Resolutions adopted by the House of Delegates

Resolution 1-76 - Descriptive Index

IT WAS MOVED AND CARRIED TO DEFER ACTION ON APPOINTING THE SPECIAL COMMITTEE UNTIL SUCH TIME AS THE FTC ACTIONS REGARDING RELATIVE VALUE SCHEDULES HAVE BEEN SETTLED.

Resolution 2-76 - Staff Privileges and Professional Liability Insurance

No further action needed.

Resolution 5-76 - Mandatory Membership in AMA - Poll

The Board approved the statement entitled "Why Unified Membership Should Be Maintained".

The Board discussed the two statements submitted by Dr. Schaller and Dr. Hyde speaking against unified membership.

IT WAS MOVED AND CARRIED TO USE THE STATEMENT AS SUBMITTED BY DR. SCHALLER AND TO EXPRESS OUR APPRECIATION TO DR. HYDE FOR HIS INTEREST EXPLAINING THAT ONLY ONE STATEMENT FOR AND ONE STATEMENT AGAINST UNIFIED MEMBERSHIP WILL BE USED.

It was pointed out that the poll will be going to all licensed physicians and it was determined to print the poll sheet going to non-members on a different color paper than the one going to members so that statistics can be developed on the basis of members and non-members.

IT WAS MOVED AND CARRIED TO APPROVE THE FORMAT OF THE PROPOSED POLL THE UNDERSTANDING THAT THE PUB—LISHING COMMITTEE WILL DEVELOP THE QUESTIONS REGARDING **ARIZONA MEDI—CINE.**

Resolution 6-76 - Establishment of a Medical Injury Commission

Referred to Malpractice Insurance Crisis Committee.

Resolution 9-76 - 1977 Budget

Last two resolves referred to Publishing Committee.

Resolution 12-76 - Housestaff Contracts and Collective Bargaining

No further action required.

Resolution 13-76 - Social Security Number

AMA delegation has been advised of our position.

Resolution 14-76 - ArMA Dues

Letter forwarded to AMA on 5/27/76.

IT WAS MOVED AND CARRIED TO SET THE FOLLOWING PAYMENT SCHEDULE FOR THE 1977 DUES - FIRST PAYMENT OF $125.00 WILL BE DUE ON JANUARY 1st AND DELINQUENT ON FEBRUARY 15. THE SECOND PAYMENT OF $125.00 WILL BE DUE ON APRIL 1st AND DE—LINQUENT ON JUNE 15th.

Resolution 17-76 - Medicaid

Recent legislature actions precludes further action.

Resolution 18-76 - Ratification of Loan

Required certification documents signed and forwarded to legal counsel for proper filing on 5/26/76 - No further action needed.

Resolution 19-76 - Discrimination in Placement Service Bulletin

Corrective instructions given 5/3/76.

Resolution 23-76 - Recognition of the Efforts of the Governor and the 32nd Legislature

Letters sent

Resolution 24-76 - Continuation of Malpractice Insurance Crisis Committee and Special Assessment

IT WAS MOVED AND CARRIED TO DO THE BILLING FOR THE SPECIAL $100 ASSESSMENT ON SEPTEMBER 1, 1976, BUT TO INVITE CONTRIBUTIONS FROM NON-MEMBER PHYSICIANS AS SOON AS FEASIBLE.

Resolution 25-76 - Relationship of ArMA to the Corporate Body of Blue Shield

The Blue Shield Board of Directors will review proposed bylaw changes on 6/23/76. The Corporate body will probably meet during late July or August to act on the proposed changes.

Resolution 27-76 - Unnecessary Air Travel Expense

IT WAS MOVED AND CARRIED TO REIM—BURSE MEMBERS WHO TRAVEL BY AIR ON ASSOCIATION BUSINESS IN AN AMOUNT EQUAL TO COACH RATES.

Resolution 29-76 - Commendation of efforts of attorneys in Malpractice Insurance Crisis

No further action needed.

Resolution 30-76 - Individually initiated Resolutions

Referred to Articles of Incorporation and Bylaws Committee on 5/27/76.

Resolution 31-76 - Commendation of Senior AMA Delegate - Seymour I. Shapiro, M. D.

No further action needed.

BOMEX APPOINTMENT

Governor Castro's letter of 5/17/76 acknowledging receipt of our nomination of Richard L. Dexter, M. D. for reappointment was received for information.

Check Signing Procedure

IT WAS MOVED AND CARRIED TO CHANGE THE CHECK SIGNING PROCEDURE SO THAT THE EXECUTIVE DIRECTOR AND THE TWO ASSOCIATE EXECUTIVE DIRECTORS ARE ADDED TO THE SIX OFFICERS WHO ARE CURRENTLY AUTHORIZED TO SIGN CHECKS. IN ADDITION, THE EMPLOYEE BOND IS TO BE INCREASED TO $100,000.00. IN ADDITION, A LIST OF ALL CHECKS WRITTEN WILL BE SUBMITTED, AT LEAST MONTHLY, TO THE TREASURER AND MEMBERS OF THE FINANCE COMMITTEE.

EXECUTIVE COMMITTEE

Classification Changes Approved

Maricopa County

(a) Peter M. Cole, M. D. - Active to Service - Account Military - One quarter dues Effective 5/25/76.
(b) Herman J. Roodman, M. D. - Active to Associate - Account Illness - Dues Exempt Effective 1/1/76.

Pima County

(a) Margaret E. Belt, M. D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/76.
(b) Louis A. Brunsting, M. D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/76.
(c) Wesley S. Fee, M. D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/76.
(d) Richard C. Finn, M. D. - Active to Affiliate - Account Left State - Dues Exempt - Effective 1/1/76.
(e) Thomas F. Griffin, Jr., M. D. - Active to Affiliate - Account Left State - Dues Exempt - Effective 1/1/76.
(f) William M. Hindman, M. D. - Active to Affiliate - Account Left State - Dues Exempt - Effective 1/1/76.
(g) William C. Nemeth, M. D. - Active to Affiliate - Account Left State - Dues Exempt - Effective 1/1/76.

Requests for Dues Refund

IT WAS MOVED AND CARRIED TO APPROVE THE REQUEST OF COUNTY MEDICAL SOCIETY TO REFUND ONE HALF YEARS DUES FOR ROY WHITE, M. D. AND FRANCISCO N. CANTU, M. D.

Request for dues exemption

IT WAS MOVED AND CARRIED TO APPROVE THE COUNTY MEDICAL SOCIETY'S REQUEST TO EXEMPT HORACE S. FUSON, M. D. FROM PAYING 1976 DUES. ACCOUNT FINANCIAL HARDSHIP.

FINANCE COMMITTEE

Financial Statement for period ending 5/31/76

Received for information.

IT WAS MOVED AND CARRIED TO CHANGE THE CURRENT CONSOLIDATED TWO PAGE STATEMENT SO THAT EACH BOARD MEMBER WILL RECEIVE THE FULL SEVEN PAGE REPORT FOR THE ACCOUNTING PERIOD PRIOR TO THE BOARD MEETING ALONG WITH A NARRATIVE PROVIDED BY THE EXECUTIVE DIRECTOR.

Borrowing Agreement

IT WAS MOVED AND CARRIED TO AUTHORIZE A BORROWING AGREEMENT IN THE AMOUNT OF $300,000.00 IN THE FORM PRESCRIBED BY THE VALLEY NATIONAL BANK.

Mutual Insurance Company of Arizona

Mr. Robinson reported that $203,555.00 had been paid back on the million dollar loan as of 5/31/76. That $2,033.20 had been paid on the Service Agreement through 4/30/76 and that $7,400.00 has been repaid on the advanced organizational costs as of 4/30/76. He also reported that as of 6/4/76; 491 applications have been received from M. D.'s and that rates have been filed with the Insurance Commissioner for hospitals, osteopaths and podiatrists.

GOVERNMENTAL SERVICES COMMITTEE

The letter from the Maricopa County Medical Society and the accompanying letter from Kenneth E. Johnson, M. D. urging that governmental hospitals be included in the P.S.R.O. program. It was pointed out that one of the AMA's proposed amendments to the PSRO laws calls for providing for PSRO review of care delivered through federal programs such as the Veterans Administration and Public Health Service. Dr. Cloud reported on current status of the AMA amendment and volunteered to obtain current information on this matter.

GRIEVANCE COMMITTEE

The AMA's letter of May 21, 1976 acknowledging receipt of the Pima County Board of Mediation report was received.

HEALTH MANPOWER COMMITTEE

The following motion was submitted to the Board for it consideration.

IT WAS MOVED AND CARRIED THAT THE SECTION ON RURAL AND MIGRANT HEALTH FORWARD A RECOMMENDATION THAT THE ARIZONA MEDICAL ASSOCIATION URGE THE ARIZONA CONGRESSIONAL DELEGATION TO SUPPORT ELIMINATION OF INEQUITIES IN CHARGES FOR HEALTH SERVICES BETWEEN RURAL AND URBAN AREAS AS PRESENTED IN S 3205, AND ENCOURAGE SENATOR TALMADGE IN HIS EFFORTS REGARDING SUCH INEQUITIES.

It was pointed out that S3205 is a tremendously long and complicated piece of legislation, some of which is not acceptable to the profession.

IT WAS MOVED AND CARRIED TO TAKE NO ACTION ON ANY PORTION OF S 3205 UNTIL THE ENTIRE BILL CAN BE ANALYZED BY THE GOVERNMENTAL SERVICES COMMITTEE

MALPRACTICE INSURANCE CRISIS COMMITTEE

Auditor's Report

The auditor's report on the income and expenditures of the 1975 assessment for the period 4/26/75 through 5/15/76 was presented as follows:

INCOME		
Assessments Collected		$191,100.00
EXPENSE		
Legal	$74,156.07	
Meetings	2,427.04	
Miscellaneous	55.38	
Printing	21,403.98	
Salaries	6,987.50	
Supplies	164.13	
Travel	3,296.09	
Consultation fees	45,355.50	
Public relations	20,916.84	
		174,762.53
EXCESS OF INCOME OVER EXPENSE at 5/15/76		$16,337.47

It was pointed out that this report was sent to all members in a Medical Memo on 6/9/76.

Appointment Confirmation

IT WAS MOVED AND CARRIED TO CONFIRM THE FOLLOWING APPOINTMENTS:

*Jack E. Brooks, M. D., Chairman
*Hayes Caldwell, M. D.
***William E. Crisp, M. D., Secretary
*Donald E. Howland, M. D.
***John F. Kahle, M. D., President-Elect
*Vernor F. Lovett, M. D.
**James F. Martin, M. D.
*Arthur R. Nelson, M. D.
***Edward Sattenspiel, M. D., President
*Donald F. Schaller, M. D.
*Richard W. Switzer, M. D.

Code: *Reappointments
**New Appointments
***Ex-Officio per bylaws

MEDICAL EDUCATION COMMITTEE

The Board received the following three actions of the Medical Education Committee for information. No action required.

1. IT WAS MOVED AND CARRIED THAT GOOD SAMARITAN HOSPITAL OF PHOENIX BE GRANTED CONTINUATION OF ACCREDITATION FOR THEIR CONTINUING MEDICAL EDUCATION PROGRAM FOR A PERIOD OF TWO YEARS AND THAT THE PROGRAM BE REEVALUATED AT THAT TIME.
2. IT WAS MOVED AND CARRIED THAT HEALTH MAINTENANCE ASSOCIATES SHOULD BE GRANTED ACCREDITATION OF THEIR CONTINUING MEDICAL EDUCATION ACTIVITIES FOR A PERIOD OF TWO YEARS WITH A RE-SURVEY TO BE CONDUCTED AT THAT TIME.
3. IT WAS MOVED AND CARRIED THAT THE VETERANS ADMINISTRATION HOSPITAL, PHOENIX, BE GRANTED ACCREDITATION OF THEIR CONTINUING MEDICAL EDUCATION PROGRAM FOR A PERIOD OF TWO YEARS WITH A RE-SURVEY TO BE CONDUCTED AT THAT TIME.

PROFESSIONAL COMMITTEE

Influenza Immunization Program

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION ENDORSE THE INFLUENZA IMMUNIZATION PROGRAM BEING IMPLEMENTED BY THE DEPARTMENT OF HEALTH SERVICES, PROMISING FULL COOPERATION WITH THE DEPARTMENT AND THE COUNTY HEALTH DEPARTMENTS: FURTHER, TO ENCOURAGE FULL COOPERATION OF ALL MEMBER PHYSICIANS IN THE PROGRAM BY VACCINATING PRIVATE PATIENTS AND RENDERING PUBLIC SERVICE AND TO EMPHASIZE THAT THERE CAN BE NO FEE CHARGED FOR THE VACCINE AS IT IS BEING PROVIDED AT GOVERNMENT EXPENSE (HOWEVER, A FEE MAY BE CHARGED FOR TIME AND MATERIAL UTILIZED IN THE VACCINATION.)

The Professional Committee recommended that Alan Gordon, M. D. be appointed as the liaison to the Department of Health Services in the implementation of the immunization program. Dr. Gordon represented the Association in the nationwide influenza immunization program briefly at the Center for Disease Control in early April and will accept such an appointment.

The following are the guidelines developed to date.

RESPONSIBILITIES FOR ADMINISTERING STATE INFLUENZA IMMUNIZATION PROGRAM

Agency	Responsibility (Functions)
Department of Health Services	1. Establish a State Planning Conference 2. Coordinate State-Wide Program and the Activities of Other State Agencies 3. Assist County Health Departments in Implementing Community Organization Activities, Including Training of Personnel 4. Develop Guidelines for Community Education and Organization 5. Develop Guidelines for Conducting Clinics 6. Develop Guidelines for Vaccine Management 7. Assess and Evaluate State-Wide Program 8. Maintain State-Wide Disease Surveillance 9. Distribute Vaccine to County Health Departments 10. Provide Laboratory Support Where Needed, and Staff Assistance 11. Coordinate and Arrange Immunization Activities for other State Agencies (e.g. Department of Corrections, MR Facilities, Legislature, Capitol Area Employees)

County Health Departments	1. Direct and Coordinate County Program 2. Arrange for Immunization Clinics, Utilizing Community Resources 3. Assess and Evaluate County Program 4. Maintain County-Wide Disease Surveillance 5. Distribute Vaccine to Local Physicians, Industries, Health Clinics, Schools, Etc. 6. Maintain "Hot Line" Desk to Answer Questions on Influenza, Clinics, High Risk Groups
Medical and Osteopathic Societies/Physicians	1. Immunize Personal Patients 2. Volunteer to Provide Medical Backup During Community Clinics 3. Collaborate in Community Education and Organization Activities 4. Collaborate in Mass Media Announcements 5. Disseminate Information on Other Immunizable Diseases 6. Report Cases of Influenza During Surveillance Period
Health Care Institutions/ Private Industries/ Schools	1. Provide Facilities for Immunization 2. Provide Immunizations 3. Disseminate Information on Other Immunizable Diseases 4. Report Absenteesim Rate and Cases of Influenza During Surveillance Period
Nurse Association/ Union Representatives/ Military Personnel/PTA/ Service Clubs/Church Groups/ Shopping Centers/ Other Voluntary Organizations and Civic Groups	1. Provide Volunteer Assistance at Clinics 2. Give Immunizations 3. Collect Patient Data 4. Disseminate Information on Health Care and Other Immunizable Diseases 5. Provide Facilities for Immunization

AMA DELEGATION REPORT

Dr. Cloud announced that Seymour I. Shapiro, M.D. will chair a reference committee at the 125th Annual Convention of the AMA in Dallas. He also noted that Dir. Shapiro has been nominated to the Council on Constitution and Bylaws.

CORRESPONDENCE

AMA letter 4/21/76

The subject letter from Bond L. Bible, Ph.D. thanking ArMA for its help during the 29th National Conference on Rural Health was received.

Burton S. Barr letter 4/27/76

The subject letter expressing thanks for our support of the continuation of the Motor Vehicle Emission Inspection Program was received.

OTHER BUSINESS

Medicaid

Dr. Howard Hyde's letter of 5/3/76 suggesting that ArMA create a substitute for Medicaid was reviewed. No action.

Malpractice Classifications

Dr. Dudley raised the problem of family physicians who do limited obstetrics being moved up to class 5 instead of being carried as class 2. It was pointed out that the insurance company deals in insurance risk factors and that according to a recent National Association of Insurance Commissioners study, "the highest awards are made for those injuries arising out of obstetrical procedures".

Dr. Dudley was urged to bring the matter to the Board of Trustees of MICA.

Meeting adjourned 11:58 a.m.

William E. Crisp, M.D.
Secretary

THE ARIZONA MEDICAL ASSOCIATION, INC.

PUBLISHING COMMITTEE

The meeting of the Publishing Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, AZ on Saturday, June 17, 1976 convened at 7:24 P.M., John W. Kennedy, M.D., Chairman and Editor presiding.

FINANCIAL STATUS OF ARIZONA MEDICINE

Mr. Robinson, at the request of the Chairman, reviewed the income and expense experience of *Arizona Medicine* for the past six years (1970-1975) based on the detailed annual auditors reports which are summarized as follows:

Year	Income*	Total Expense	Operating Loss
1975	$26,024.00	$64,168.00	$(38,144.00)
1974	30,179.00	56,306.00	(26,127.00)
1973	37,350.00	51,768.00	(14,418.00)
1972	42.239.00	52.937.00	(10,698.00)
1971	42.302.00	54.594.00	(12,292.00)
1970	$34,856.00	$50,463.00	$(15,607.00)

*This figure does not include that portion of the dues that is applied to the Journal operations as a subscription fee.

It was pointed out that two things have occurred, advertising revenues have dropped and printing costs have skyrocketed. As an example, in 1970, it cost $36,598.16 to print 1,198 pages, in 1975 it cost $48,925.00 to print 1,050 pages. Non-printing costs rose only $1,376.16 between 1970 and 1975.

For the first five months of 1976 non-dues revenue was $9,022.69, while operating expenses was $29,304.55. That for the year 1976, non-dues revenue was projected at $30,300.00 with expenses projected at $69,700.00 for a projected operating loss of $39,400.00.

With trend of declining revenue and increasing printing costs, the 1977 budget projected non-dues revenue of $21,400.00 and operating expenses of $72,935.00.

ADVERTISING/EDITORIAL RATIOS

Year	Adv. Pages	Adv. %	Edit. Pages	Edit. %	Total Pages	Total %
1970	418	35%	780	65%	1198	100%
1971	461	47%	523	53%	984	100%
1972	515	51%	485	49%	1000	100%
1973	457	49%	468	51%	925	100%
1974	374	36%	652	64%	1026	100%
1975	356	34%	694	66%	1050	100%

Mr. Robinson reviewed the above ratios pointing out that, generally speaking, in order for money to made without the help of a portion of the members' dues being applied to the revenue of the journal, 60% of the journal must be directed to advertising.

LOCAL AND NATIONAL ADVERTISING PROMOTIONS

The several attempts of developing more local advertising using agencies, direct mail and our own commissioned agent were reviewed. None of these attempts have been more than moderately successful.

The history of our relations with the national advertising agencies were reviewed. Prior to 1965 we used State Medical Journal Advertising Bureau, Inc. (SMJAB) in Chicago. From 1965 through 1975 we used State Journals West which disbanded. We have returned to SMJAB who advise that the trend has been, and continues to be, for the national advertisers to use the national "throw away" books instead of the state association sponsored books. The current fee paid to SMJAB is 17% + 2% discount.

PRINTER

Mr. Robinson reviewed the history of our relations with the printers of *Arizona Medicine* from the first issue in January 1944, which was printed by the A. J. Taylor Printing Company, to the present.

RESOLUTION 9-76 AND POLL

Dr. Sattenspiel reviewed the background of Resolution 9-76, specifically those two resolves dealing with *Arizona Medicine* which read as follows:

> "RESOLVED, That a poll of all licensed physicians practicing in Arizona be conducted, in conjunction with the poll on unified membership, regarding *Arizona Medicine*, its composition, suggested changes and its continuance; and be it further
>
> RESOLVED, That the Board of Directors be directed to consider the pripriety of a continuing $50,000 per year loss to the Arizona Medical Association in the publication of *Arizona Medicine* and prepare an appropriate resolution for introduction at the 1977 House of Delegates meeting."

Extensive discussion ensued following with these conclusions:

1. That Marshall B. Block, M.D. and Warren Gorman, M.D. would prepare a statement about *Arizona Medicine* should be continued, several questions dealing with readership of specific sections would be included. Also to be included would be the opportunity to make recommendations on how to improve *Arizona Medicine*.

Although other aspects of the journal operation were discussed, it was determined to delay further actions until the results of the poll were available for the committee.

Meeting adjourned 8:41 p.m.

William E. Crisp, M.D.
Secretary
by
Bruce E. Robinson
Executive Director

ArMA
Medical History

VETERANS ADMINISTRATION HOSPITAL

PHOENIX, ARIZONA

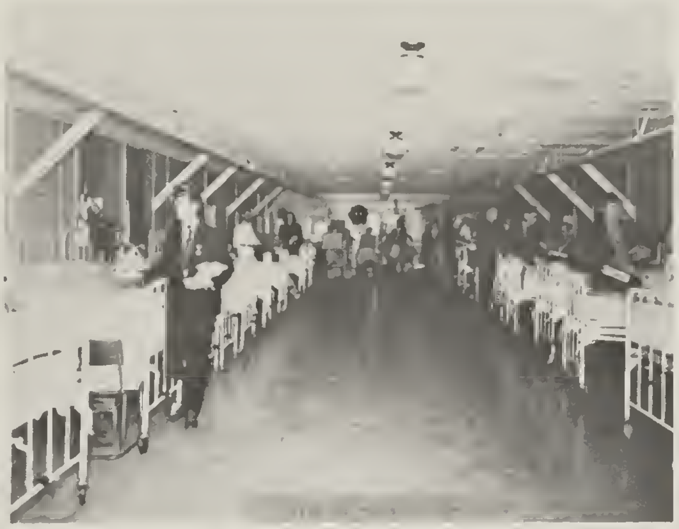
The present site of the hospital at 7th Street and Indian School Road is, as some of the long time residents will recall, not the original site.

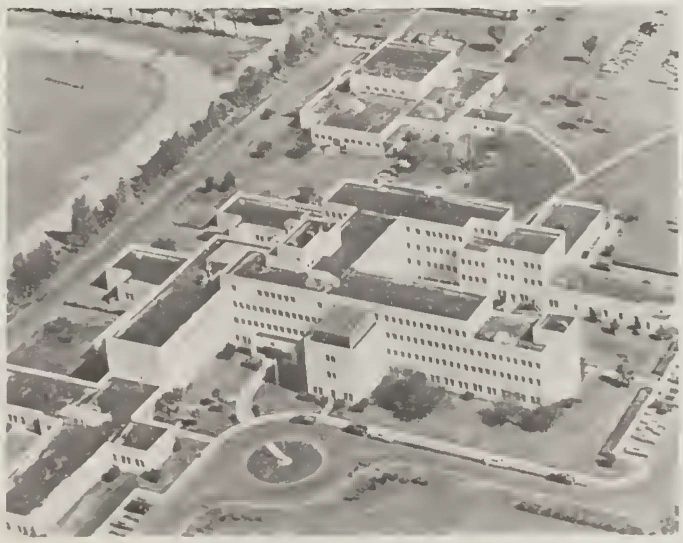
It was first established in June, 1946 at which time the hospital of a prisoner of war camp at Papago Park, was converted for use by the V.A.

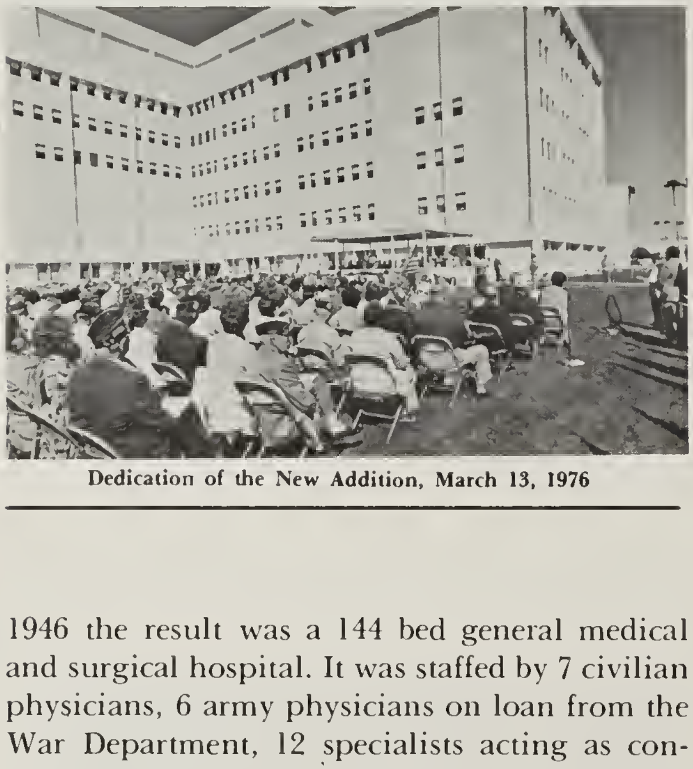
This Papago site had been a prisoner of war camp. As many as 3,000 German POW's were detained. The history of this camp itself is exciting and dismal. It was the site of escape by tunneling underneath the fence and was the site of the murder of an inmate by seven of his fellow German prisoners. This led to "the last mass execution in the United States" they were hanged at Fort Leavenworth, Kansas on August 24, 1945 just after the war had ended in Europe, even some of the military thought this was somewhat unwarranted considering the nature of the original crime and the circumstances under which it occurred. But that is another story.

The VA Hospital at 7th & Indian School Road was started on August 8th, 1949 and completed July 2, 1951. And as you know another addition at the same site has now been added. This is quite an imposing structure and on the inside has wide spacious halls, many waiting rooms and even some beds. The hospital hand-out states that the original structure was for a 200 bed capacity and this was to be increased by 328 beds with this last addition.

To return to the original installation at Papago Park, it is stated "public officials, private citizens, and various service organizations protested the army's decision to close the encampment and successfully persuaded the Veterans Administration to locate a much needed hospital on the site." Groups such as the Boy Scouts, Navy Mothers, Elks, and Fraternal Order of Policemen helped with the renovation. In


Christmas, 1947, at the Papago Park Hospital.


Aerial View (looking northwest) of the Newly Completed Hospital at Seventh Street and Indian School Road, 1952.



Dedication of the New Addition, March 13, 1976

1946 the result was a 144 bed general medical and surgical hospital. It was staffed by 7 civilian physicians, 6 army physicians on loan from the War Department, 12 specialists acting as consultants and 32 nurses. Today the hospital has 55 physicians, 220 nurses, and a total of 770 persons on the payroll. The first year of operation resulted in nearly 2,300 patients treated at a cost of $781,000. Near the end of the first year President Truman signed an agreement transferring land from the Department of the Interior to the VA. This made it possible to start the process of constructing a permanet hospital at the corner of 7th Street & Indian School Rd., and this was done at a cost of $5,000,000 and the 192 bed hospital was completed and dedicated September 9, 1951. Dr. Seymour Fisher was the director of the hospital from 1951 to 1960."

The following statistics are furnished by the Veterans Facility and they state that nearly 5,900 inpatients will be treated this year at a cost of $15,000,000. The dental service will have 4,200 sittings for treatment. There will be nearly 100,000 outpatient visits. Volunteers will contribute 70,000 hours of service to the hospital. The pharmacy service will mail out 120,000 prescriptions statewide. The per diem cost which includes salaries for the physicians, is $115.00. So if you are impressed with the veterans facility medical costs, we should all love and look forward to a national health insurance plan. The length of patient stay is not stated.

Further information furnished by the facility states that the hospital provides many of the latest services, these include nuclear medicine, respiratory therapy, renal dialysis, unit dose, an outpatient clinic, a dental clinic, a new one-half million dollar automatic transport system (I wonder what it transports), a consolidated laundry serving Prescott, Tucson and Phoenix, an eye ear nose and throat clinic, rehabilitation medicine and intensive care units.

The present chief of staff is Dr. H. D. Bouman and the director is Milton Michael, Jr.

REFERENCES

The Phoenix Veterans Administration Hospital, Past, Present & Future by William D. Esson, Administrative Resident.
Veterans Administration Hospital 328 bed addition dedication program - March 13, 1976.
Marshall Justice, the Last Mass Execution in the United States by Richard Whittingham; Henry Regnery Company Chicago - 1971.

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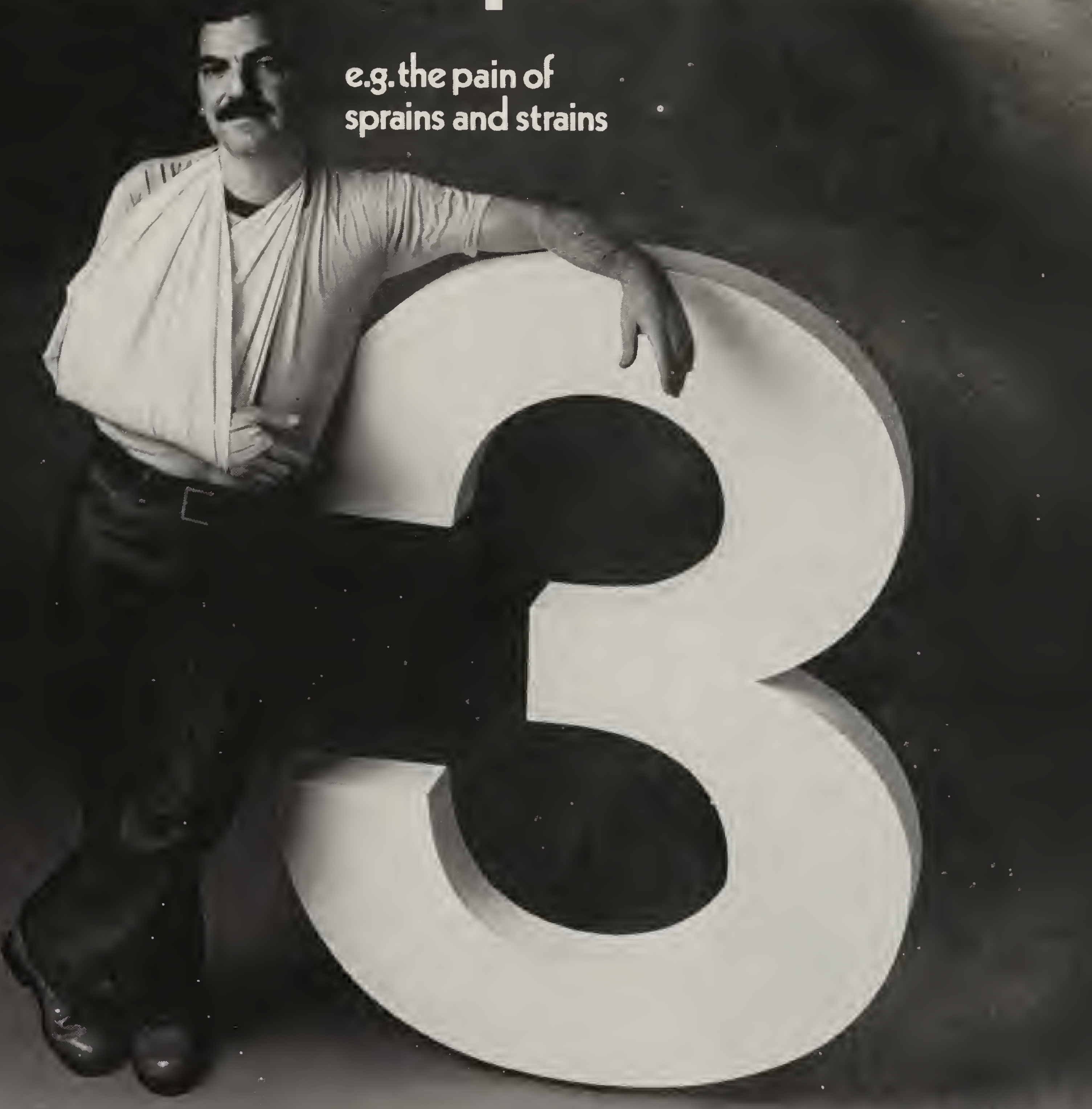

e.g. the pain of
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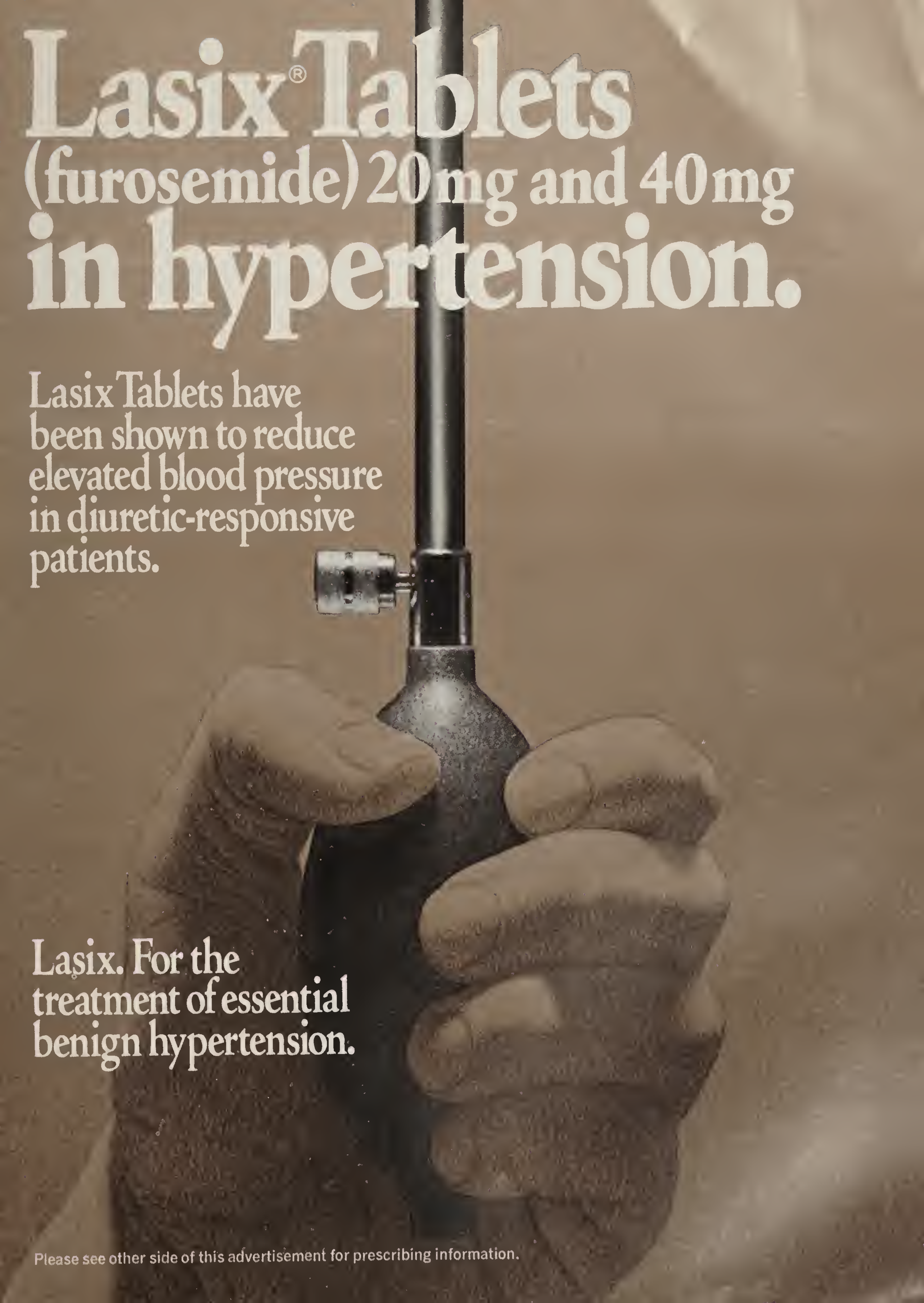

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Lasix Tablets have been shown to reduce elevated blood pressure in diuretic-responsive patients.

Lasix. For the treatment of essential benign hypertension.

Please see other side of this advertisement for prescribing information.

Lasix® Tablets
(furosemide) 20 mg and 40 mg
in hypertension.

A brief summary of the Prescribing Information for
Lasix® (furosemide) Tablets 20 mg and 40 mg

WARNING—Lasix (furosemide) is a potent diuretic which if given in excessive amounts can lead to a profound diuresis with water and electrolyte depletion. Therefore, careful medical supervision is required, and dose and dose schedule have to be adjusted to the individual patient's needs. (See under "Dosage and Administration.")

Indications—Lasix (furosemide) is indicated for the treatment of the edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Hypertension—Lasix (furosemide) may be used for the treatment of hypertension alone or in combination with other antihypertensive drugs. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controllable with Lasix (furosemide) alone.

CONTRAINDICATIONS—Because animal reproductive studies have shown that Lasix (furosemide) may cause fetal abnormalities, the drug is contraindicated in women of childbearing potential. (See "Additional Information.")

Lasix (furosemide) is contraindicated in anuria. If increasing azotemia and oliguria occur during treatment of severe progressive renal disease, the drug should be discontinued. In hepatic coma and in states of electrolyte depletion, therapy should not be instituted until the basic condition is improved or corrected. Lasix (furosemide) is contraindicated in patients with a history of hypersensitivity to this compound.

Warnings—Excessive diuresis may result in dehydration and reduction in blood volume, with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients. Excessive loss of potassium in patients receiving digitalis glycosides may precipitate digitalis toxicity. Care should also be exercised in patients receiving potassium depleting steroids.

Frequent serum electrolyte, CO_2 and BUN determinations should be performed during the first few months of therapy and periodically thereafter, and abnormalities corrected or the drug temporarily withdrawn.

In patients with hepatic cirrhosis and ascites, initiation of therapy with Lasix (furosemide) is best carried out in the hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma; therefore, strict observation is necessary during the period of diuresis. Supplemental potassium chloride and, if required, an aldosterone antagonist are helpful in preventing hypokalemia and metabolic alkalosis.

Patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

In those instances where potassium supplementation is required, an oral liquid preparation should be used rather than enteric-coated potassium salts.

There have been several reports, published and unpublished, concerning nonspecific small-bowel lesions consisting of stenosis, with or without ulceration, associated with the administration of enteric-coated thiazides with potassium salts. These lesions may occur with enteric-coated potassium tablets alone or when they are used with nonenteric-coated thiazides, or certain other oral diuretics.

These small-bowel lesions have caused obstruction, hemorrhage, and perforation. Surgery was frequently required, and deaths have occurred.

Available information tends to implicate enteric-coated potassium salts, although lesions of this type also occur spontaneously. Therefore, coated potassium-containing formulations should be administered only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Patients with known sulfonamide sensitivity may show allergic reactions to Lasix (furosemide).

Precautions—As with any potent diuretic, electrolyte depletion may occur during therapy with Lasix (furosemide), especially in patients receiving higher doses and a restricted salt intake. Electrolyte depletion may manifest itself by weakness, dizziness, lethargy, leg cramps, anorexia, vomiting, and/or mental confusion.

Asymptomatic hyperuricemia can occur and gout may rarely be precipitated. Reversible elevations of BUN may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

When parenteral use of Lasix (furosemide) precedes its oral use, it should be kept in mind that cases of tinnitus and reversible hearing impairment have been reported. There have also been some reports of cases in which irreversible hearing impairment occurred. Usually, ototoxicity has been reported when Lasix (furosemide) was injected rapidly in patients with severe impairment of renal function at doses exceeding several times the usual recommended dose and in whom other drugs known to be ototoxic were often given. If the physician elects to use high dose parenteral therapy in patients with severely impaired renal function, controlled intravenous infusion is advisable (for adults, it has been reported that an infusion rate not exceeding 4 mg Lasix [furosemide] per minute has been used).

Increases in blood glucose, and alterations in glucose tolerance tests with abnormalities of the fasting and two-hour postprandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Lasix (furosemide) may lower serum calcium levels, and rare cases of tetany have been reported.

Patients receiving high doses of salicylates, in conjunction with Lasix (furosemide) may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Diuretics such as furosemide may enhance the nephrotoxicity of cephaloridine. Therefore, Lasix (furosemide) and cephaloridine should not be administered simultaneously.

Sulfonamide diuretics have been reported to decrease arterial responsiveness to pressor amines and to enhance the effect of tubocurarine. Great caution should be exercised in administering curare or its derivatives to patients undergoing therapy with Lasix (furosemide), and it is advisable to discontinue Lasix (furosemide) for one week prior to any elective surgery.

Adverse Reactions—Various forms of dermatitis, including urticaria and rare forms of exfoliative dermatitis, erythema multiforme, pruritus, paresthesia, blurring of vision, postural hypotension, nausea, vomiting, or diarrhea.

Anemia, leukopenia, aplastic anemia, and thrombocytopenia (with purpura). Rare cases of agranulocytosis which responded to treatment.

In addition, the following rare adverse reactions have been reported; however, relationship to the drug has not been established with certainty: sweet taste, oral and gastric burning, paradoxical swelling, headache, jaundice, thrombophlebitis and emboli and acute pancreatitis.

Lasix (furosemide)-induced diuresis may be accompanied by weakness, fatigue, lightheadedness or dizziness, muscle cramps, thirst, increased perspiration, urinary bladder spasm, and symptoms of urinary frequency.

Dosage and Administration

ADULTS

The usual adult dose of Lasix (furosemide) is 20 to 80 mg given as a single dose.

If the diuretic response with a single dose of 20 to 80 mg is not satisfactory, the following schedule should be used: Increase this dose by increments of 20 or 40 mg not sooner than 6 to 8 hours after the previous dose until the desired diuretic effect has been obtained. This individually determined single dose should then be given once or twice daily. The dose of Lasix (furosemide) may be carefully titrated up to 600 mg per day in those patients with severe clinical edematous states.

With doses exceeding 80 mg/day and given for prolonged periods, careful clinical and laboratory observations are particularly advisable.

Hypertension—The usual dose of Lasix (furosemide) is 40 mg twice daily both for initiation of therapy and for maintenance. Careful observations for changes in blood pressure must be made when this compound is used with other antihypertensive drugs, especially during initial therapy. The dosage of other agents must be reduced by at least 50 percent as soon as Lasix (furosemide) is added to the regimen to prevent excessive drop in blood pressure. As the blood pressure falls under the potentiating effect of Lasix (furosemide), a further reduction in dosage, or even discontinuation, of other antihypertensive drugs may be necessary. It is further recommended, if 40 mg twice daily does not lead to a clinically satisfactory response, to add other hypotensive agents, e.g., reserpine, rather than to increase the dose of Lasix (furosemide).

INFANTS AND CHILDREN

Pediatric Administration: The usual initial dose of oral Lasix in infants and children is 2 mg/kg body weight, given as a single dose. If the diuretic response is not satisfactory after the initial dose, dosage may be increased by 1 or 2 mg/kg not sooner than 6 to 8 hours after the previous dose. Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

How Supplied—Lasix Tablets 40 mg (furosemide) supplied as white, round, monogrammed, scored tablets.

Lasix Tablets 20 mg (furosemide) supplied as white, oval, monogrammed tablets.

Note: Dispense in dark containers. Exposure to light may cause slight discoloration which, however, does not alter potency.

Additional Information

Toxicology

The acute toxicity of Lasix (furosemide) has been determined in mice, rats, and dogs. In all three animal species, the oral LD_{50} of Lasix (furosemide) exceeded 1000 mg/kg of body weight, while the intravenous LD_{50} ranged from 300 to 680 mg/kg. Intragastric injection of the drug in newborn rats resulted in an LD_{50} of 380 mg/kg.

The acute toxicity of high doses of Lasix (furosemide) was characterized by convulsions, paralysis, and collapse. Surviving animals often became dehydrated and depleted of electrolytes due to the diuresis induced by Lasix (furosemide). In the newborn rats, intragastric injection of the drug caused hyperactivity and anorexia.

Chronic toxicity studies with Lasix (furosemide) were done in rats and dogs. In a one-year study in rats, renal tubular degeneration occurred, with all doses higher than 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day). A six-month study in dogs revealed calcification and scarring of the renal parenchyma at all doses above 10 mg/kg (83 percent of the maximal recommended human dose of 600 mg per day).

Reproductive Studies

The effects of Lasix (furosemide) on embryonic and fetal development and on pregnant dams were studied in mice, rats, and rabbits.

Lasix (furosemide) caused unexplained maternal deaths and abortions in the rabbit when 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day) was administered between days 12 to 17 of gestation. In a previous study the lowest dose of only 25 mg/kg (2 times the maximal recommended human dose of 600 mg per day) caused maternal deaths and abortions. In a third study, none of the pregnant rabbits survived a dose of 100 mg/kg. Data from the above studies indicate fetal lethality which can precede maternal deaths.

The results of the mouse study and one of the three rabbit studies also showed an increased incidence of hydronephrosis (distention of the renal pelvis and, in some cases, of the ureters) in fetuses derived from treated dams as compared to the incidence in fetuses from the control group.

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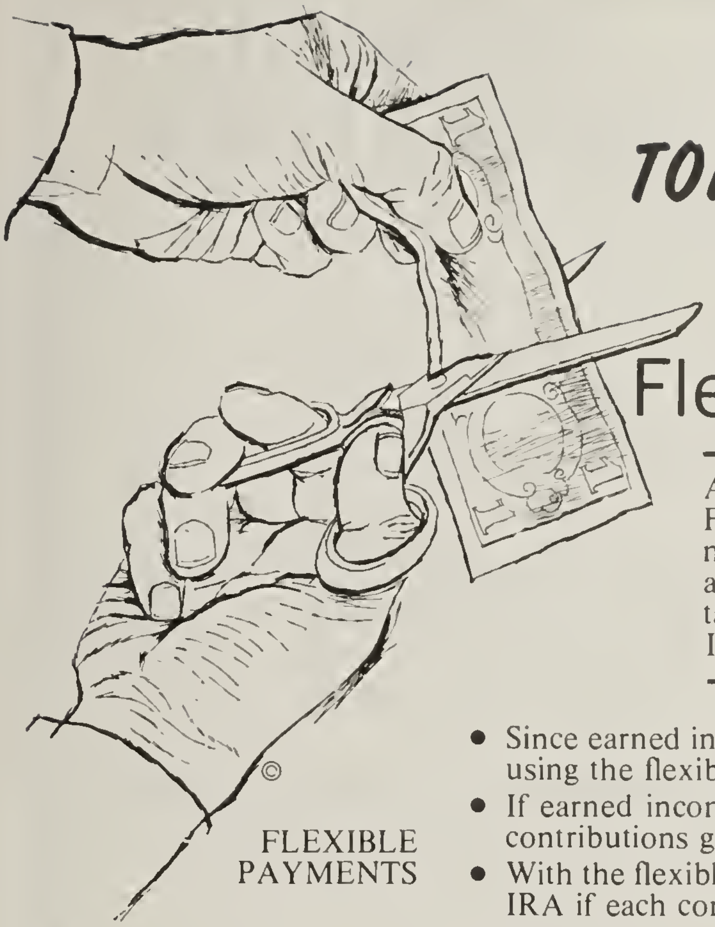

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			Male	Female	Male	Female
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45	32,164	45,264	205	186	361	337
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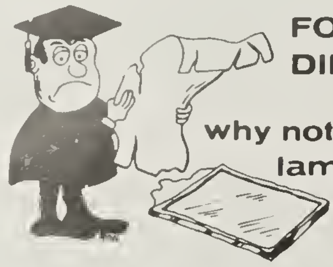

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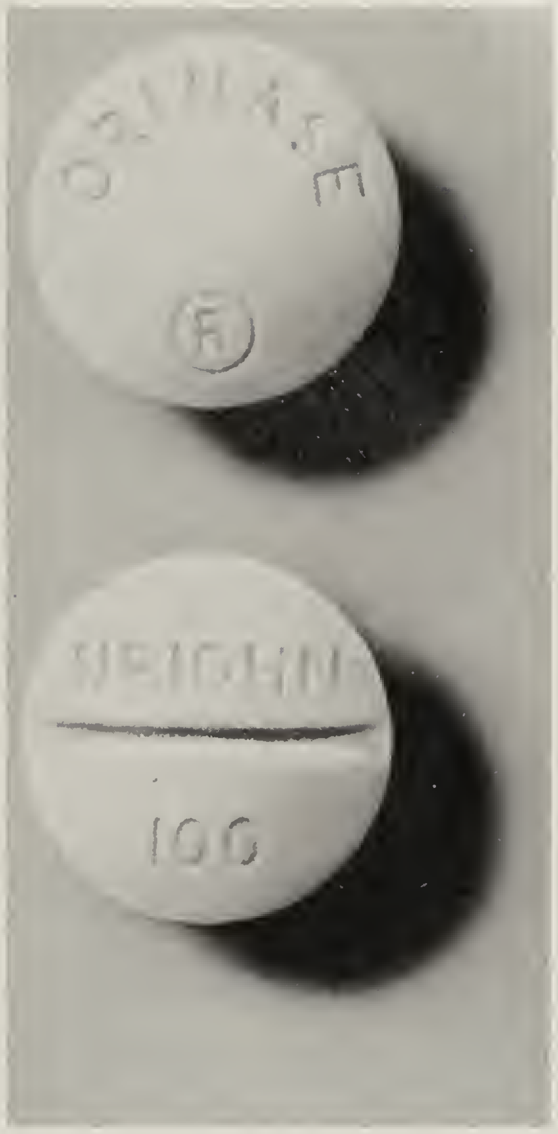

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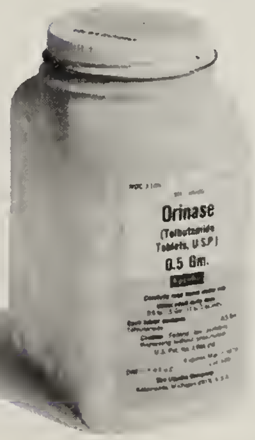

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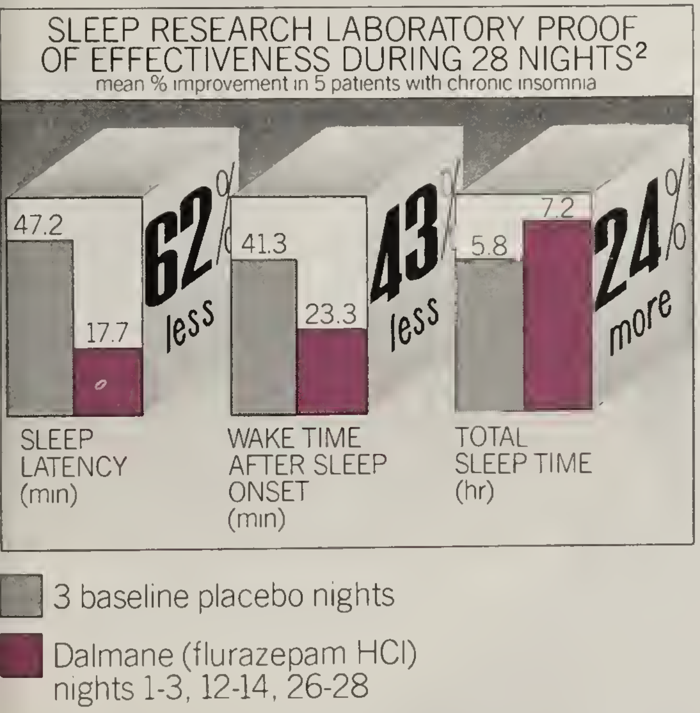
For insomnia that is a chronic problem...

Only Dalmane (flurazepam HCl) offers sleep laboratory proof of effectiveness for as long as 28 nights

Continued relief of insomnia in patients with chronic insomnia

Since insomnia is often transient and intermittent, the prolonged administration of a hypnotic is generally not necessary or recommended. But when insomnia is a chronic or recurring problem, continued effectiveness is as important as initial effectiveness. Results of a recently published sleep research laboratory study[1] demonstrated that, while pentobarbital lost effectiveness within two weeks, Dalmane maintained effectiveness for 28 consecutive nights. Similar 28-night results with Dalmane, displayed below, were obtained by a second sleep research group.[2] In previous studies,[3] both chloral hydrate and glutethimide began to lose effectiveness after several nights, while Dalmane maintained effectiveness throughout the 14 medication nights. Whether the problem is difficulty falling asleep, staying asleep or sleeping long enough, consider these results when selecting a sleep medication.



Patient benefits include relative safety, infrequent morning "hang-over"

Dalmane is well tolerated, seldom causing morning drowsiness or grogginess.[4] No increase in dosage is required for continued effectiveness from night to night.[1-3] Should Dalmane be used repeatedly, periodic blood counts and liver and kidney function tests should be performed. The usual adult dose is 30 mg *h.s.*, but 15 mg may suffice for some patients and is recommended as a starting dose for the elderly and debilitated to help preclude oversedation, dizziness or ataxia.

Continued relief of insomnia: One more good reason to specify

Dalmane®
(flurazepam HCl)

One 30-mg capsule *h.s.*— usual adult dosage (15 mg may suffice in some patients).
One 15-mg capsule *h.s.*— initial dosage for elderly or debilitated patients.

whenever a hypnotic is needed

ROCHE

Please see following page for a summary of product information.

New objective proof: continued insomnia relief without increasing dosage...[1,2]

Dalmane®

(flurazepam HCl) ⒾⓋ

Objectively proved in the sleep research laboratory...

during 28 consecutive nights of administration:

☐ **effectiveness with a single 30-mg *h.s.* dose, maintained**

☐ **rapid sleep induction, maintained**

☐ **sleep for 7 to 8 hours, on average, maintained**

☐ **less time awake during the night, maintained**

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; and in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.

Contraindications: Known hypersensitivity to flurazepam HCl.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Use in women who are or may become pregnant only when potential benefits have been weighed against possible hazards. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated, initial dosage should be limited to 15 mg to preclude oversedation, dizziness and/or ataxia. If combined with other drugs having hypnotic or CNS-depressant effects, consider potential additive effects. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported were headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase. Paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, have also been reported in rare instances.

Dosage: Individualize for maximum beneficia effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES:

1. Kales A, *et al*: *Clin Pharmacol Ther* *18*:356-363, Sep 1975
2. Dement WC, *et al*: Long-term effectiveness of flurazepam 30 mg *h.s.* on chronic insomniacs. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, Jun 30-Jul 4, 1975
3. Kales A, *et al*: *Arch Gen Psychiatry* *23*:226-232, Sep 1970
4. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ

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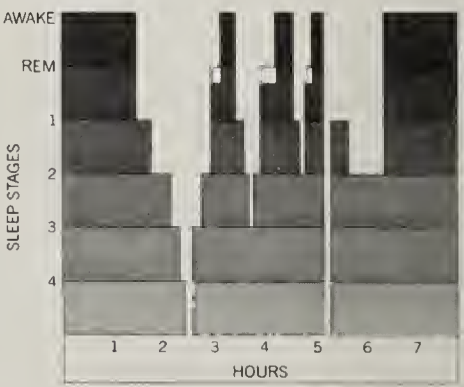

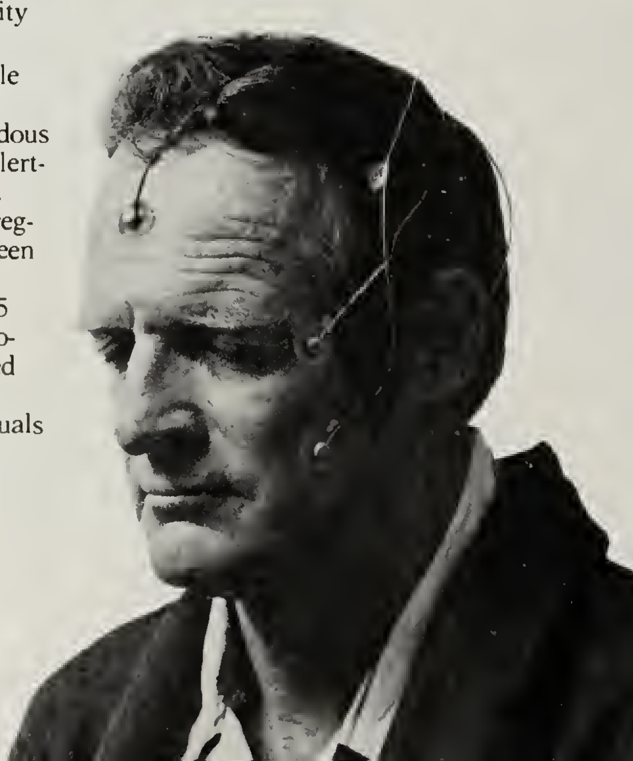

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Holiday Inn

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CONTACT:
Robert I. Cutts, M.D.
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CONTACT: Irwin M. Freundlich, M.D.
Arizona Medical Center
Dept. of Radiology
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Phoenix, AZ 85033

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CONTACT:
Howard Kimball, M.D.
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1201 South 7th Avenue
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D-5 North Conference Rm., Good Samaritan Hospital, Phoenix, AZ

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CONTACT:
Bernard E. Levine, M.D.
Pulmonary Function Laboratory
Good Samaritan Hospital
1033 E. McDowell Hospital
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CONTACT:
Stuart M. Gould, Jr., M.D.
Medical Director
Camelback Hospital
5055 N. 34th St.
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Dir. Medical Education
7300 E. 4th St.
Scottsdale, AZ

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CLINICAL PATHOLOGICAL CONFERENCE

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3rd Wednesday Every Month
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Good Samaritan Hospital, Phoenix, AZ

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926 E. McDowell Road
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4TH TUESDAY OF EACH MONTH

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CONTACT:
A. L. Forte, M.D.
St. Mary's Hospital
Tucson, AZ 85724

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3rd Monday of each month 7:30 a.m.
Room 4410, Arizona Medical Center, Tucson, AZ

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College of Medicine

CONTACT:
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Arizona Medical Center
Dept. of Surgery
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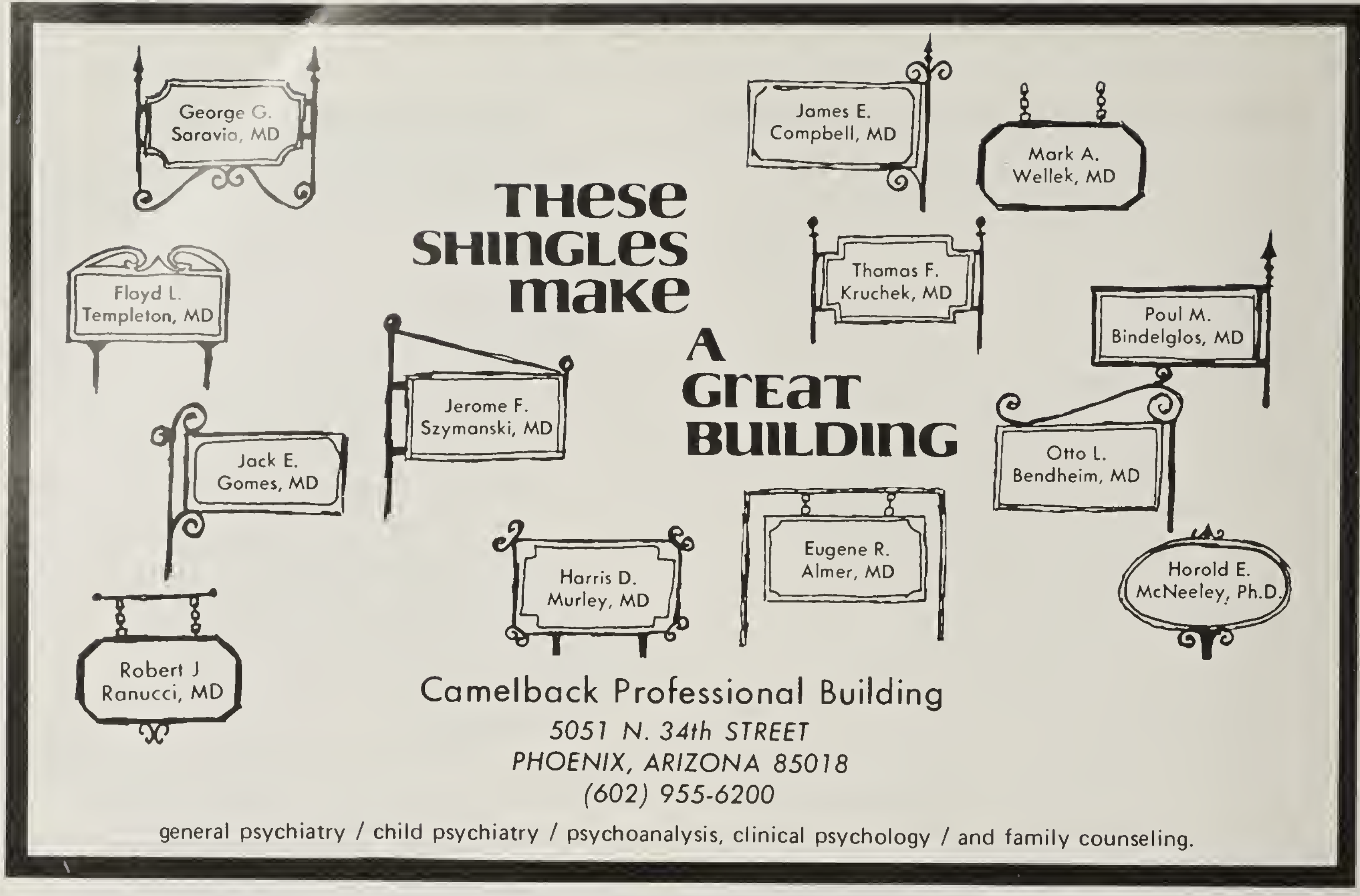

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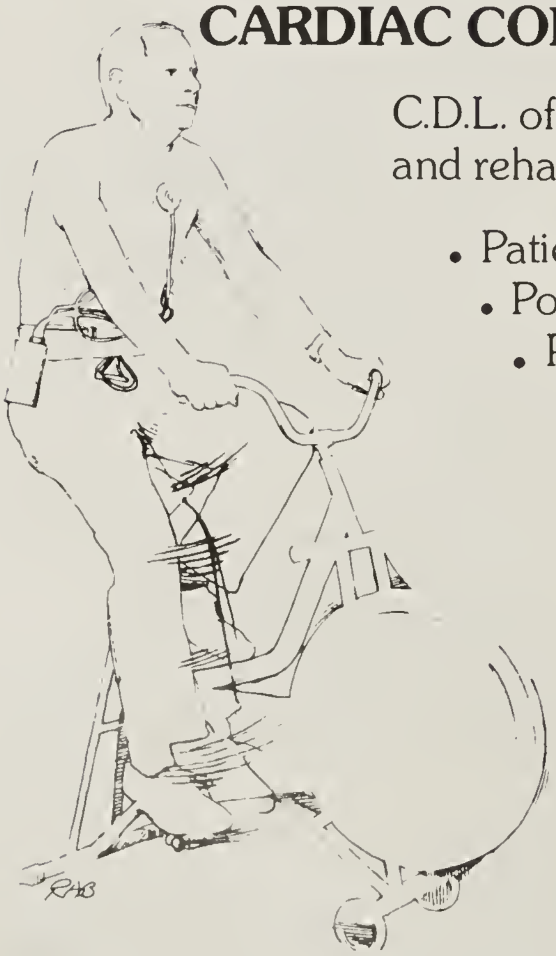

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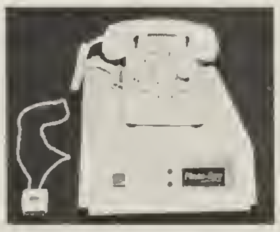

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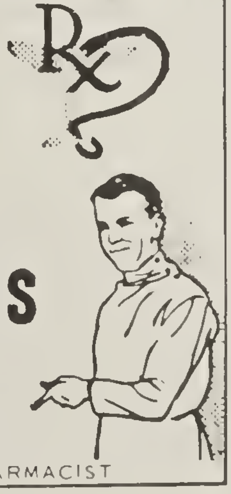

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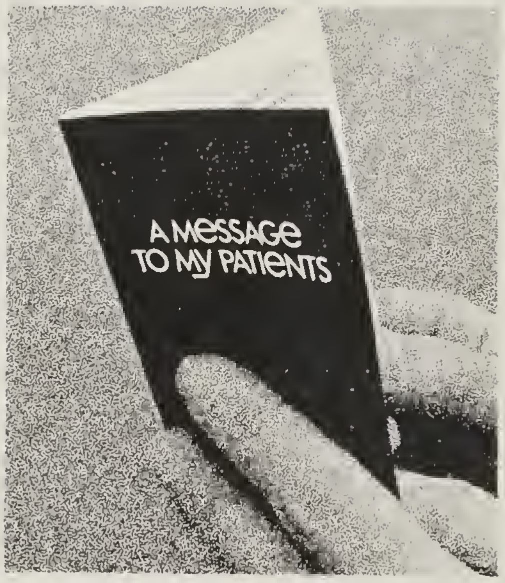

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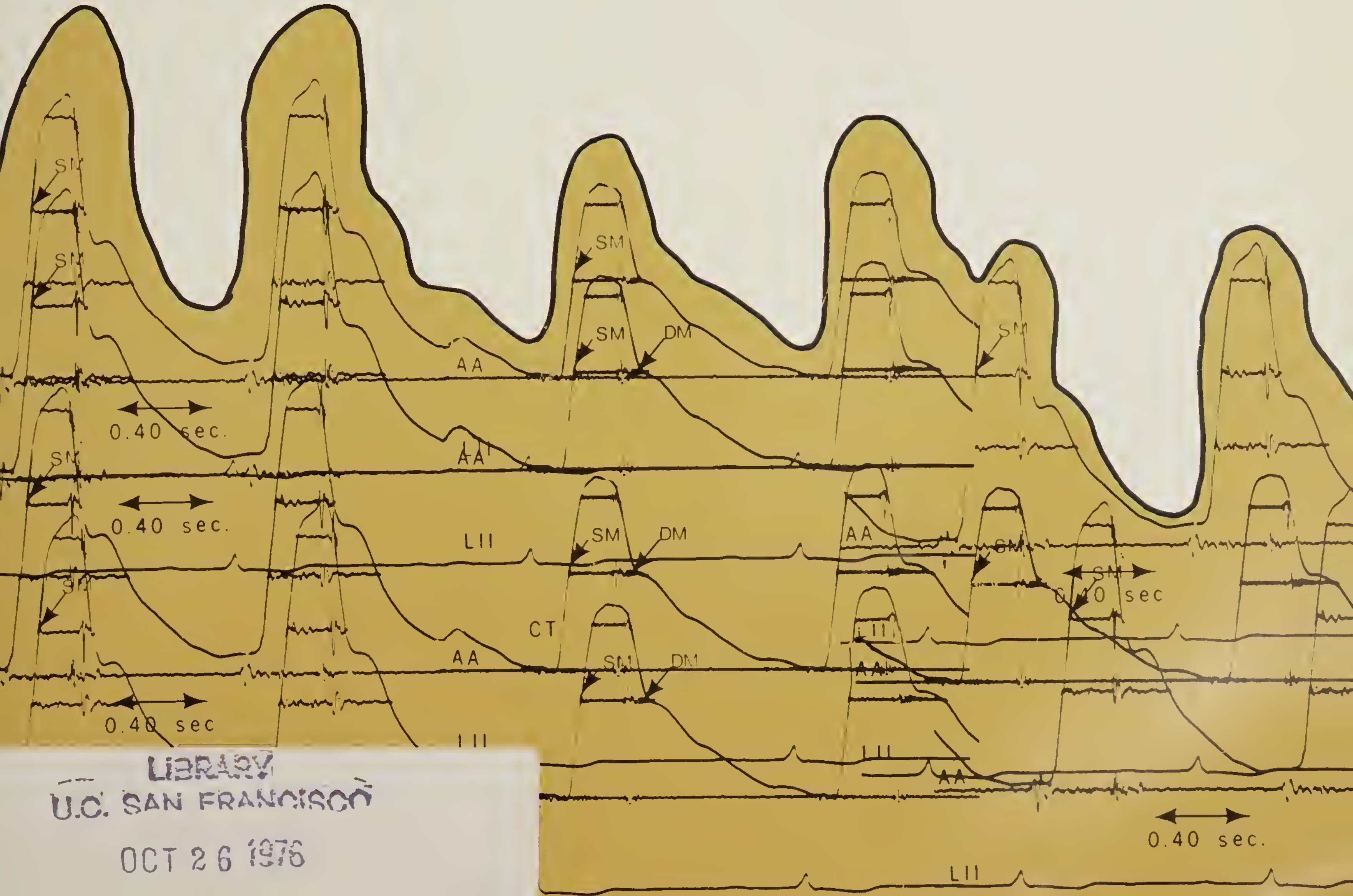

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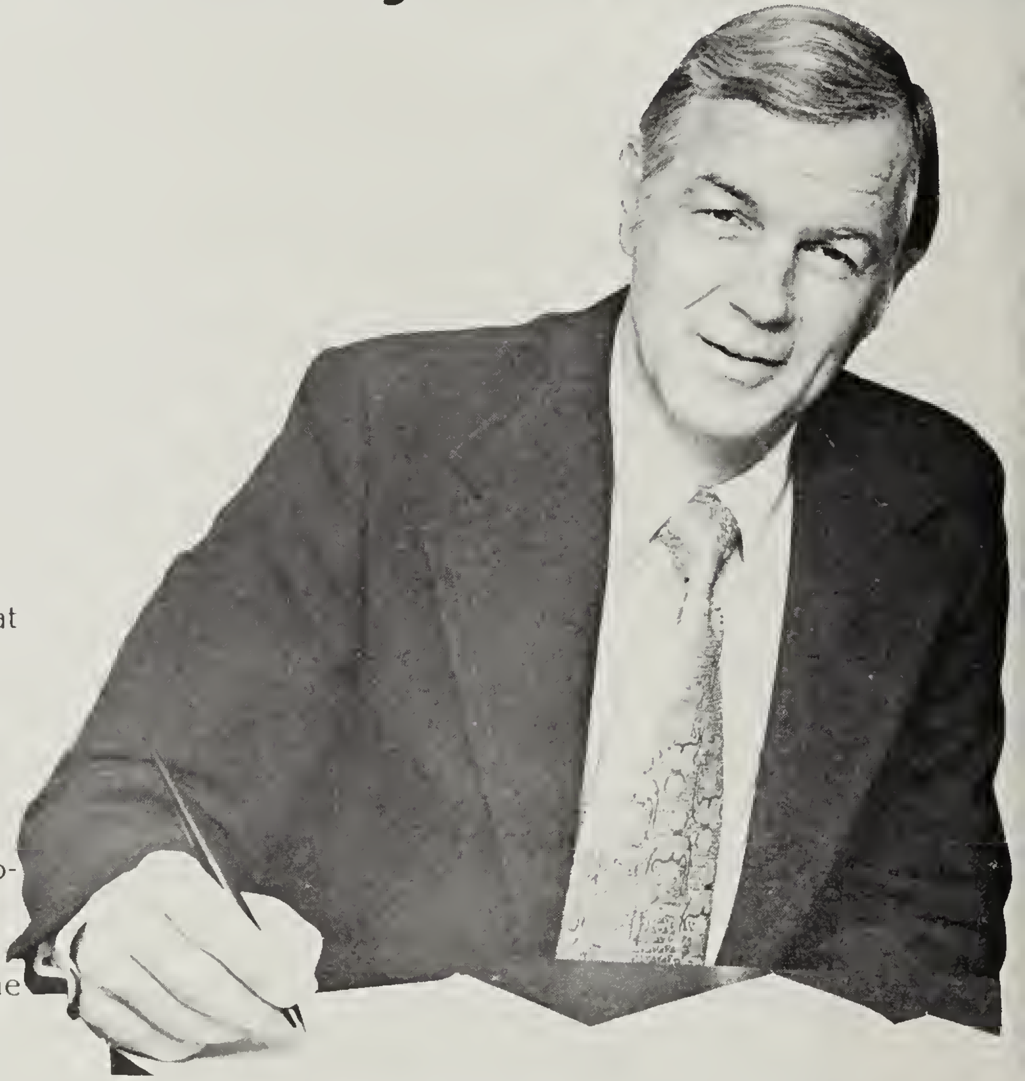

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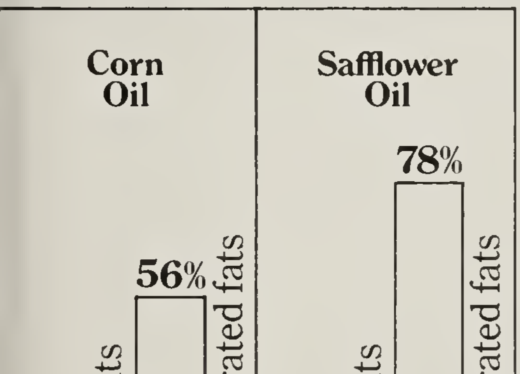

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*INDICATIONS. Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

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Meclizine HCl is contraindicated in individuals who have shown a previous hypersensitivity to it.

WARNINGS. Since drowsiness may, on occasion, occur with use of this drug, patients should be warned of this possibility and cautioned against driving a car or operating dangerous machinery.

Usage in Children: Clinical studies establishing safety and effectiveness in children have not been done; therefore, usage is not recommended in the pediatric age group.

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ADVERSE REACTIONS. Drowsiness, dry mouth and, on rare occasions, blurred vision have been reported.

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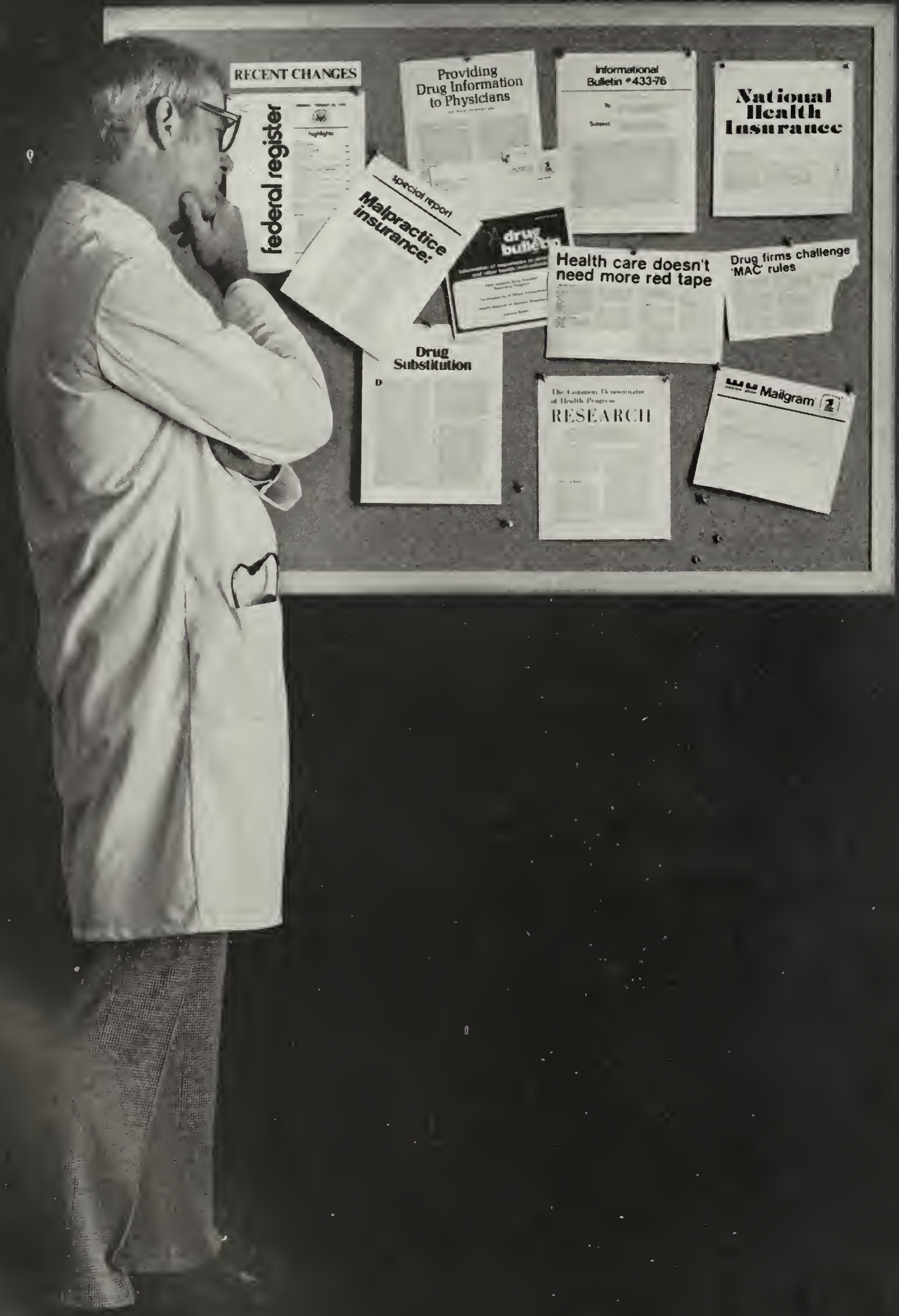
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THERE ARE A LOT OF PEOPLE GETTING BETWEEN YOU AND YOUR PATIENT.

Medicine today is in the spotlight, subjected to all kinds of scrutiny. Your control over patient therapy is being monitored, judged and occasionally abrogated, sometimes by unknown third parties.

The worry is that in the wake of this focus, the relationship between you and your patient will be weakened, without offsetting benefits. Consider three examples:

Drug substitution In most states, pharmacy laws, regulations or professional custom stipulate that your non-generic prescriptions be filled with the precise products you prescribe. But in the last five years, a dozen or more State laws have been changed, permitting the pharmacist in most cases to select a product of the same generic drug to fill any prescription.

Ironically, this dilution of physician control has taken place against a background of growing evidence that purportedly equivalent drug products may be inequivalent, since neither present drug standards nor their enforcement are optimal. In fact, the FDA itself says it has not enforced the same standards for hundreds of "follow-on" products that it had applied to the original NDA approvals. Thus physician control over patient therapy is being eroded with a risk that patients may be exposed to drugs of uncertain quality.

The major advertised claim for substitution is reduced prescription prices for consumers. Yet no documentation of any significant savings has been produced.

MAC Maximum Allowable Cost, MAC for short, is a Federal regulation designed to cut the Government's drug bill by setting price ceilings for drugs dispensed to Medicare and Medicaid patients. Unless the prescriber certifies on the prescription that a particular product is medically necessary, the Government intends to pay only for the cost of the lowest-priced, purportedly-equivalent, generally-available product. The effect of the program may be that elderly and indigent patients will be restricted to products which someone in Washington believes are priced right. Practicing doctors will have little to say about administration of the program, since Government will have absolute authority to make its choices stick.

The drug lag The future of drug and device research depends upon a scientific and regulatory environment that encourages therapeutic innovations. The American pharmaceutical industry annually is spending more than $1 billion of its own funds and evaluating more than 1,200 investigational compounds in clinical research. Disease targets include cancer, atherosclerosis, viruses and central nervous system disorders, among others. But there is a major barrier to the flow of new drugs to your patients: The cost of the research is more than ten times what it was, per product, in 1962; and whereas governmental clearance of new drug applications took six months then, it commonly consumes two years now.

The FDA needs adequate time, of course, to consider data. But it is equally clear that the present approval process contributes to needless delay of needed therapy. That's why the increased efficiency of the drug approval process is vital to all our futures.

If these issues concern you, we suggest that you make your voice heard—among your colleagues and your representatives in State legislatures and in Washington.

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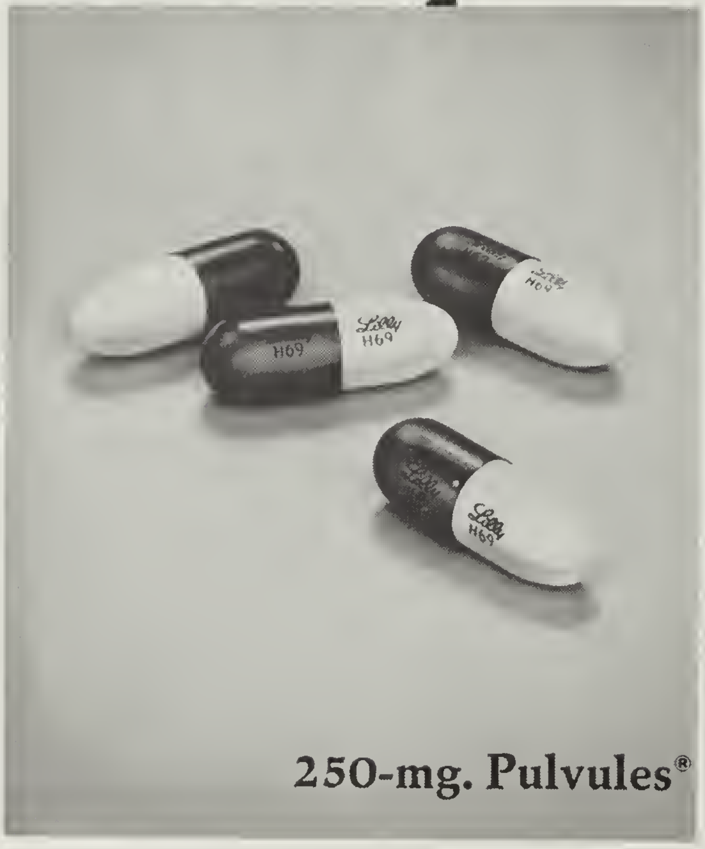

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Original Articles

PROLAPSE OF THE ATRIOVENTRICULAR VALVES AND ASSOCIATED MITRAL STENOSIS: A CASE REPORT

PAUL F. HOWARD, M.D.
ALBERTO BENCHIMOL, M.D.
KENNETH B. DESSER, M.D.

ABSTRACT — SUMMARY

A 54 year old man presented with auscultatory features of mitral stenosis and aortic insufficiency. Echocardiography demonstrated systolic prolapse of the tricuspid and mitral valves in association with diastolic mitral stenosis. Cardiac catheterization and angiocardiography were diagnostic of tricuspid and mitral valve prolapse, mitral valve stenosis and aortic insufficiency. Pathologic examination of the resected mitral valve revealed mitral stenosis, pliable mitral leaflet edges, fused thickened chordae and histologic evidence of myxomatous degeneration. It is proposed that this case represents coexisting atrioventricular valve prolapse and rheumatic heart disease. Correlation of the echocardiographic, catheterization and pathologic findings indicates the diagnostic usefulness of reflected ultrasound.

Since its initial description, mitral valve prolapse has been subject to intensive investigation.[1-6] The pathophysiologic basis of prolapse is still incompletely understood and associated clinical features may be expressed in varied fashion.[7-14] Many disease states, including rheumatic heart disease, have been implicated in the genesis of the prolapse syndrome, yet their exact relationship with floppy mitral valves is unclear.[1,7,8,11,15]

We describe here a patient who exhibited tricuspid valve prolapse in association with coexisting mitral valve prolapse and stenosis. The clinical, non-invasive, angiographic and histopathologic findings in this case provide unique features which have not been previously documented.

CASE REPORT

A 54 year old man was referred to the Institute for Cardiovascular Diseases with a heart murmur. The patient had two documented episodes of acute rheumatic fever associated with acute migratory polyarthritis as a young adult. He was subsequently refused military service because of abnormal cardiac auscultation. For the 10 years prior to admission he experienced progressive exertional dyspnea which was treated with digoxin and diuretics.

At the time of admission the blood pressure was 130/70, heart rate 68/min, respirations 14/min, and temperature 37°C. The peripheral pulses were bounding in nature and the point of maximal cardiac pulsation was in the sixth intercostal space at the left anterior axillary line. Auscultation at the mitral area revealed a loud first heart sound, normal second heart sound, opening snap and low pitched diastolic rumble. A Grade II/VI systolic ejection murmur was heard at the aortic, tricuspid and mitral areas. An arterial decrescendo diastolic murmur was heard at the 3rd intercostal space to the left of the sternum. These findings were confirmed on a phonocardiogram, (Figure 1). No systolic clicks were audible at the mitral or tricuspid areas with the patient in the supine, left lateral decubitus, sitting, or standing positions. The rest of the physical examination was unremarkable.

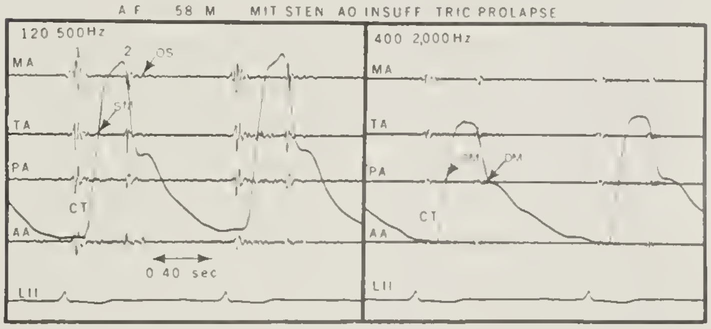

Figure 1:

Simultaneously recorded mitral (MA), tricuspid (TA), pulmonic (PA) and aortic area (AA) phonocardiograms, external carotid pulse tracing (CT) and lead II (L II) of the electrocardiogram. An opening snap (OS), systolic murmur (SM) and diastolic murmur (DM) are present.

To our knowledge this report represents the first description of tricuspid valve prolapse in association with combined mitral valve prolapse and stenosis. Mitral valve prolapse has been a subject of much discussion in the recent litera-

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Submitted for publication 1 76.
From: The Institute for Cardiovascular Diseases, Good Samaritan Hospital, 1033 East McDowell Road, Phoenix, Arizona 85006.
Supported in Part by the E. Nichols and Kim Sigsworth Memorial Funds and The Institute for Cardiovascular Diseases, Inc.
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ture and its cause and manifestations are undergoing vigorous investigation. Prolapse of the tricuspid valve into the right atrium has similarly been demonstrated to occur[10,16-22] and usually with concomitant mitral valve prolapse. The clinical hallmark of atrioventricular valve prolapse is the midsystolic click and/or late systolic murmur yet auscultation may occasionally be normal in affected subjects.[7] The patient described in this report did not have any auscultatory evidence of prolapse.

Numerous mechanisms have been advanced in order to explain mitral and tricuspid prolapse[7,12,15,23-25] and of those proposed the valvular theory appears to have the most merit.[11,13] Myxomatous degeneration of the valve leaflets has been demonstrated in patients so affected and may provide the anatomic substrate for the pathogenesis of this clinical syndrome.[7,11,26] Abnormal orientation of the papillary muscles and disordered left ventricular contraction patterns have also been implicated as possible causes of prolapse.[7,11-13,15,18] Disease states associated with the prolapse syndrome include Marfan's syndrome, Turner's syndrome, rheumatic heart disease, idiopathic hypertrophic subaortic stenosis, congenital heart disease, and coronary artery disease.[1,3,7,8,14] Most investigators believe, however, that prolapse in its "pure form" is due to degenerative changes of the mitral valve apparatus, with floppy redundant leaflets and chordae tendineae. Non-invasive and invasive data described in this report represented features common to both rheumatic valvular disease and "classical" prolapse. Ballooning of the atrioventricular valves during systole was demonstrated echocardiographically and proven by angiography. A dyskinetic portion of the anterior wall of the left ventricle was also identified. Examination of the mitral valve revealed myxomatous degeneration as well as evidence of chronic rheumatic valvular disease. The patient also exhibited a past history suggestive of acute rheumatic fever and physical findings with supportive catheterization data indicating aortic insufficiency and mitral stenosis. In fact, the presence of an apical diastolic rumble, opening snap, ejection systolic murmur and arterial diastolic murmur along with the absence of a midsystolic click dictated careful angiographic evaluation in order to confirm unsuspected but echocardiographically demonstrable bivalvular prolapse.

A resting electrocadiogram revealed sinus rhythm, left atrial enlargement, first degree A-V block, and left ventricular hypertrophy with "strain". Echocardiography was performed with an Ekoline 20 ultrasonoscope interfaced with a DR-8 Electronics for Medicine strip chart recorder. Tricuspid and mitral echograms were diagnostic of bivalvular prolapse in association with mitral stenosis. There was fluttering of the anterior mitral valve leaflet, suggesting aortic insufficiency. The left atrial internal dimension was increased, (Figures 2 and 3).

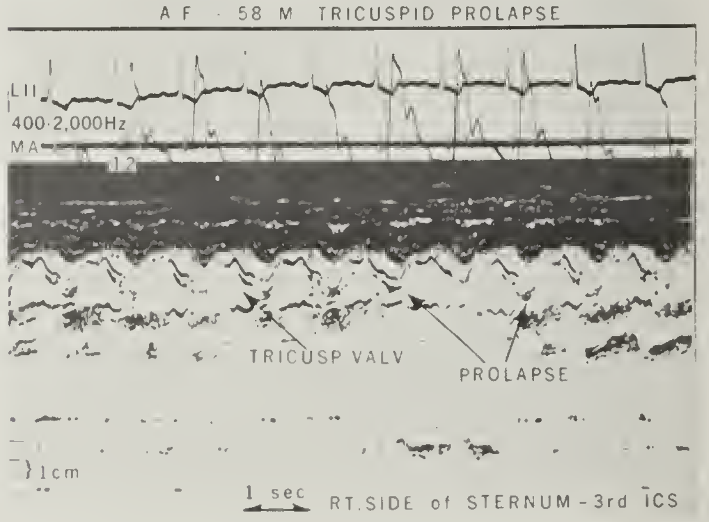

Figure 2:

Simultaneous lead II (L II) of the electrocardiogram, mitral area (MA) phonocardiogram, external carotid pulse tracing (CT) and tricuspid valve echogram recorded from the third intercostal space at the right sternal border. Note the pansystolic prolapse of the anterior leaflet of the tricuspid valve.

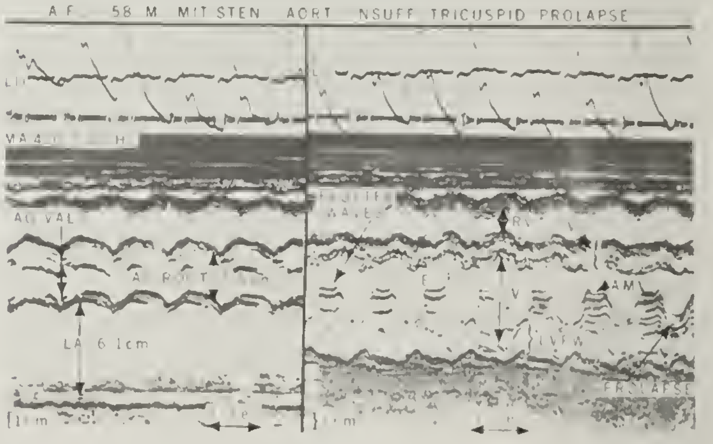

Figure 3:

Simultaneously recorded lead II (L II) of the electrocardiogram, mitral area (MA) phonocardiogram, external carotid pulse tracing and echocardiograms. Left panel: There is enlargement of the left atrial (LA) internal dimension. Right panel: The mitral valve echogram shows fine fluttering of the anterior leaflet, reduced diastolic closing velocity of the anterior leaflet (flat E-F slope), abnormal anterior motion of the posterior leaflet, pansystolic prolapse and thickening of both leaflets. (RV = right ventricle, IVS = interventricular septum, LV = left ventricle, AMV = anterior mitral valve, LVPW = left ventricular posterior wall).

Cardiac catheterization was performed in the post-absorptive state. All pressures were measured with fluid filled catheters connected to a Statham p23Db strain gauge. Right atrial, right ventricular and pulmonary atery pressures were normal. The pulmonary arterial wedge pressure was elevated to 25mmHg and a mean gradient of 15mmHg was recorded across the mitral valve. Left ventricular pressures were normal, and there was no aortic transvalvular gradient. The cardiac index was 2.2 L/min/M^2. Biplane cineangiography was performed with sodium and meglumine diatrizoate (Hypaque-M75%) and recorded on a 35mm Arriflex camera at a speed of 50 frames/sec. Right and left ventricular angiograms demonstrated prolapse of the tricuspid and mitral valves respectively, (Figures 4 and 5). Left ventriculography revealed mild anterior wall dyskinesis. There was no angiographic evidence of mitral regurgitation. Cinefluoroscopy showed calcification of the mitral valvular apparatus without involvement of the anulus. Mild aortic insufficiency was noted during an aortic root injection of dye. Selective coronary cineangiography was normal.

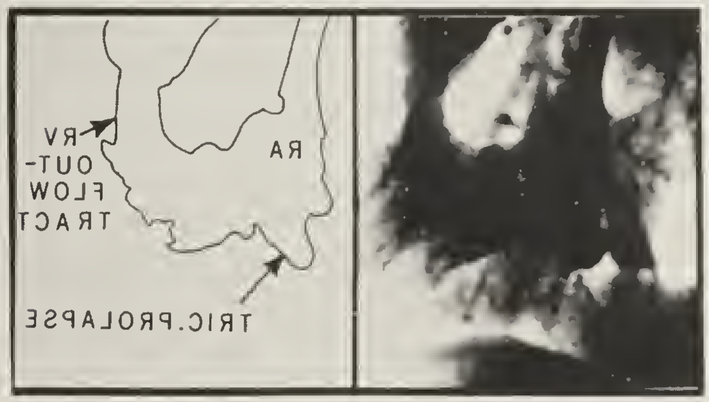

Figure 4:

Injection of radiocontrast material into the superior vena cava with follow-through right ventricular angiogram demonstrates tricuspid valve prolapse. The patient was in sinus rhythm during the entire course of the angiogram.

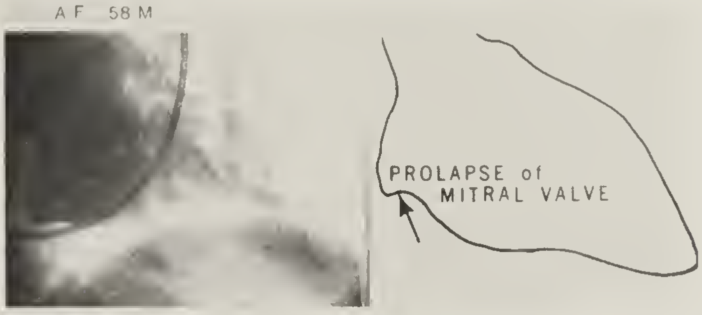

Figure 5:

Left ventricular angiogram in the right anterior oblique projection demonstrates systolic mitral valve prolapse and anterior wall dyskines's. The patient's electrocardiogram showed sinus rhythm before and during left ventriculography.

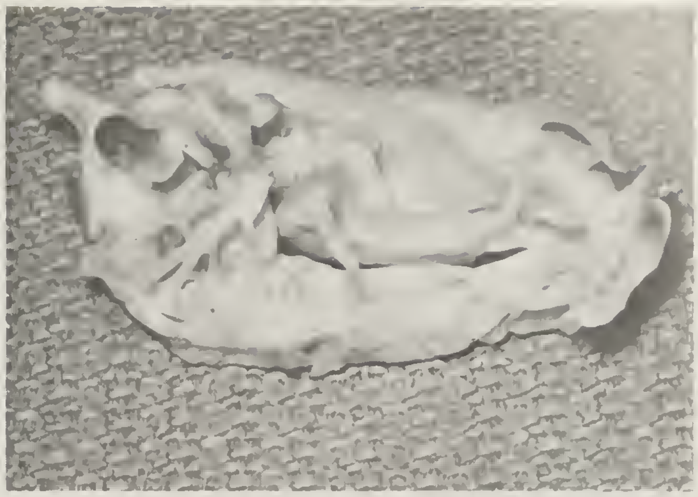

Figure 6:

Gross appearance of the resected mitral valve. Note the "fish mouth" appearance of the valvular orifice and the thickened, fused chordae tendineae.

At surgery, the mitral chordae tendineae were thickened and fused and the valve orifice had a fish mouth appearance. The valve cusps were thickened and sclerosed yet the free margins of the leaflets were pliable. The gross appearance of the resected valve is shown in Figure 6. Mitral valve replacement was accomplished with a #29 Hancock procine heterograft. Histologic examination of the removed valve demonstrated calcification of the base of the mitral valvular apparatus and histiocytic infiltration of the anulus and leaflets. Areas of myxomatous degeneration were also noted. Aschoff bodies were not present, (Figure 7).

The diversity of physical findings, non-invasive recordings and catheterization data render it difficult to ascribe all the abnormal findings to a single etiologic factor. Idiopathic prolapse has not been associated with fused commissures, shortened chordae tendineae, and stenosis of the mitral valve. Calcification of the mitral valve apparatus has been reported in subjects with prolapse but the pattern has generally been anulus calcification in the elderly patient.[7,11,26,27] On the other hand, rheumatic heart disease is not classically correlated with myxomatous degeneration of the valvular apparatus and documented cases of tricuspid valve prolapse have not been previously noted in the presence of rheumatic heart disease.[2,7,17,27,28] The patient described here exhibited evidence of left ventricular anterior wall dyskinesis, yet this phenomenon can be seen in association with both disease states.

Thus, this case provided an example of atrioventricular valve prolapse with features of rheumatic heart disease. It is impossible to

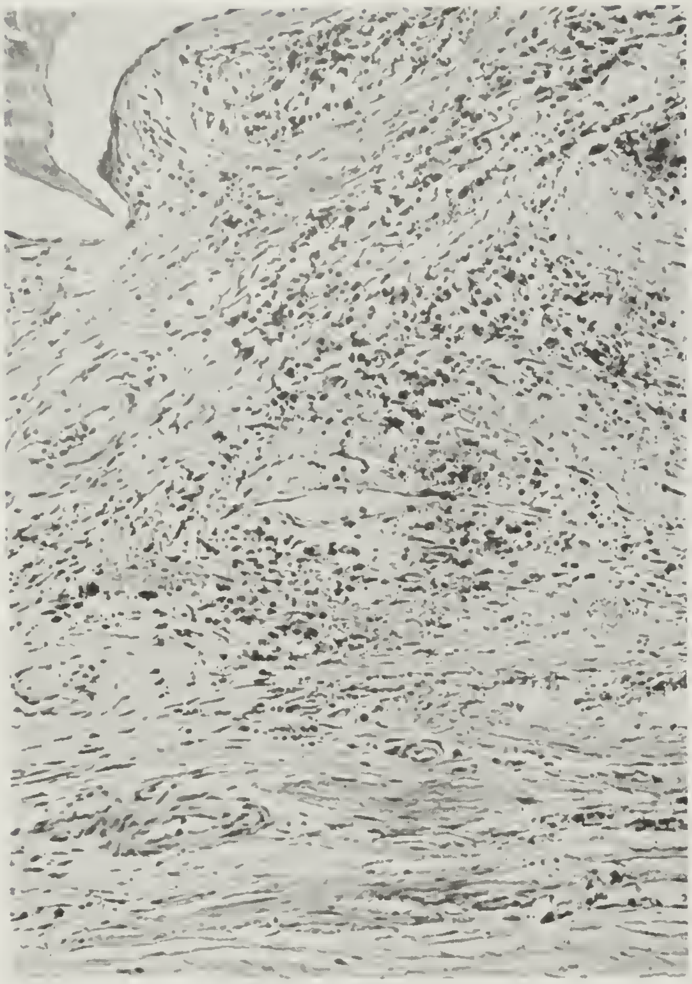

Figure 7:

Photomicrograph of valve tissue demonstrates areas of neovascularization, histiocytic infiltration and presence of myxomatous change. (X 100).

conciliate all the data on the basis of a unitary diagnostic hypothesis but there is important information to be gained from this case study. Idiopathic atrioventricular valve prolapse is currently recognized as a common disease[29 30] and a rheumatic process superimposed on a preexisting floppy mitral valve is not unlikely. The unique combination of systolic mitral valve prolapse in combination with diastolic mitral stenosis, suggested by echocardiographic examination and correlated with a pathologically proven narrowed mitral valve with pliable free leaflet margins indicates the rewarding value of ultrasonic examination.

KEY INDEX WORDS

Mitral Valve Prolapse
Tricuspid Valve Prolapse
Mitral Stenosis
Rheumatic Heart Disease
Aortic Insufficiency
Echocardiography
Phonocardiography
Auscultation

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SUBACUTE BACTERIAL ENDOCARDITIS (SBE) SUPERIMPOSED ON IDIOPATHIC HYPERTROPHIC SUBAORTIC STENOSIS (IHSS)

A Case Report With Special Emphasis On Prophylactic Antibiotic Therapy

MOHAMMAD A. HABIBZADEH, M.D.
GEORGE W. CURD, JR., M.D., FACP
NICHOLAS H. ZELLER, M.D., FACP

INTRODUCTION

Although bacterial endocarditis in patients with IHSS has previously been considered to be very rare, an incidence of about 5-10% has been reported in recent years.[P·Q] However, there is no general agreement relating to the use of prophylactic antibiotic administration at time of surgical procedures in these patients. The purpose of this report is to emphasize that because of associated patho-anatomic and hemodynamic abnormalities, patients with IHSS are particularly susceptible to bacterial endocarditis and appropriate antibiotic prophylaxis should be instituted in such patients.

CASE REPORT

A 54-year old male was in good health until about 5 months prior to admission when he developed a flu-like illness with fever and generalized malaise. Symptoms persisted in spite of 2-one week courses of Tetracycline therapy. A month before admission he was re-evaluated by his private physician and six blood cultures were obtained, all of which were positive for Alpha hemolytic Streptococci. Immediate hospitalization was advised but the patient did not comply. He continued to have daily temperature spikes of 100° to 101°, chilly sensations, anorexia, constipation, weight loss and occasional bedshaking chills with gradual deterioration of his general condition until he was admitted to the hospital.

The presence of a heart murmur was detected in his teens and when he was examined for the military service, in which he served for two years without symptoms.

On physical examination he appeared acutely ill, anemic and underweight (111 pounds - Ht. 5′8″). Temperature was 102°F. He had one small petechial hemorrhage in the right lower conjunctival sac. There was no clubbing of the fingers or skin petechiae. He had brisk and bifid peripheral pulses. The lungs were clear. Blood pressure was 100 70 mm Hg. The point of maximum cardiac impulse was over the fifth intercostal space in the midclavicular line and was moderately forceful and sustained. On auscultation, the first and second heart sounds were heard clearly. There was a loud presystolic gallop sound and a medium pitched III IV crescendo-decrescendo murmur which started after the first and ended before the second heart sound. The murmur was best heard over the fifth intercostal space,

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From: Cardiovascular Section, Veterans Administration Hospital, 7th Street and Indian School Road, Phoenix, Arizona 85012. Reprint requests to Dr. Habibzadeh.

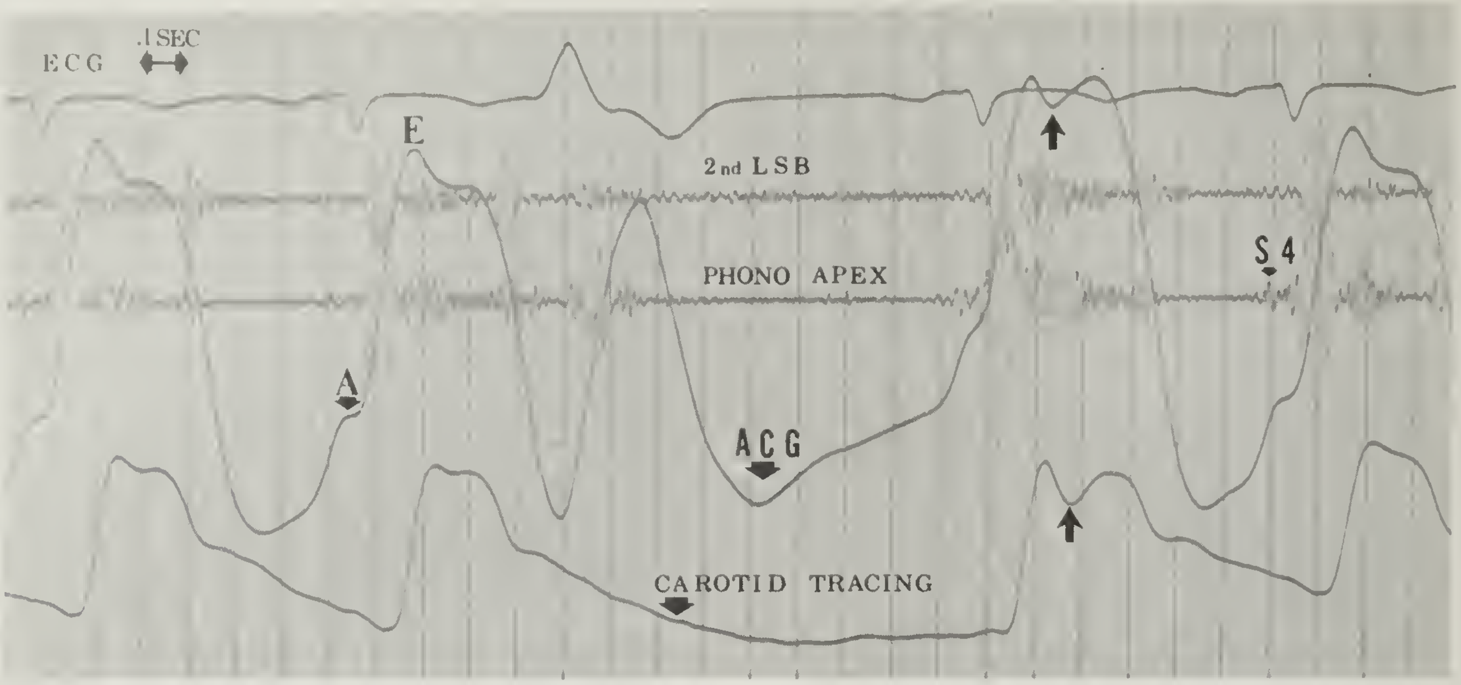

Figure 1

Simultaneously recorded electrocardiogram (ECG), Phono- and apex-cardiogram (ACG), with carotid sphygmogram, exhibit a crescendo-decrescendo systolic murmur, a very tall A wave on the ACG, a presvtolic gallop (S4), with bifid carotid and apical impulse, especially in the post-extrasystolic beat without increasing the amplitude of carotid impulse.

midway between the left midclavicular line and left sternal border, radiating to the axilla and along the left sternal border. There was no diastolic murmur. He had no hepatosplenomegaly, Oral examination revealed dental caries and moderate periodental disease.

LABORATORY DATA: On admission, the white blood count was 12,200 mm^3, neutrophils, 84%; lymphocytes, 11%; hemoglobin, 10.7 gm; hematocrit, 32.1%; erythrocyte sedimentation rate, 68%. The rheumatoid factor was positive and antinuclear factor negative. Serum protein electrophoresis, complement level and antistreptolysin 0 titer were normal. Chest X-Ray revealed no cardiomegaly and the lung fields were clear. Electrocardiogram and vectocardiogram showed regular sinus rhythm and loss of anterior forces.

Of the six blood cultures obtained within the first twenty-four hours of admission, five were positive for Alpha hemolytic Streptococci.

HOSPITAL COURSE: Twenty-four hours following admission, the patient was started on aqueous penicillin intravenously and streptomycin intramuscularly. For the first two weeks of his hospitalization, his temperature fluctuated with elevations up to 100.4° associated with a gradual drop of hemoglobin to 6.1 mg%, hematocrit to 17.7%, and reticulocyte count to 0.2%. On the fourteenth hospital day the patient developed acute thrombophlebitis of the right calf, which was successfully treated with intravenous infusion of dextran and heparin. During the third week of treatment, gradual improvement was noted. His antibiotic therapy was discontinued after the fourth week. He remained afebrile and multiple blood cultures were negative. Nine weeks after cessation of his antibotic therapy, he developed sudden onset of severe pain and blanching of the right 5th finger extending to the lateral part of his palm. It became reddish and swollen during the following week with gradual improvement thereafter. Blood cultures were negative and there was no fever or sign of infection at the site of the lesion. This was considered to be the result of an aseptic embolus to the extremity.

The patient's cardiac status was further evaluated during the convalescent period. Phonocardiogram revealed a presystolic gallop, with a clear first heart sound followed by a crescendo-decrescendo murmur ending before the second heart sound. There was no diastolic murmur. The apexcardiogram revealed a tall A-Wave measuring about one-third of apical systolic impulse, with a clear post extrasystolic accentuation of the midsystolic dip associated with augmentation of the systolic murmur. His carotid pulse tracing showed a rapid upstroke which was distinctly bifid after the extrasystoles (Fig #1). Simultaneous phonocardiogram and carotid tracing studies with Valsalva maneuver (Fig #2) and amyl nitrite inhalation (Fig #3) were

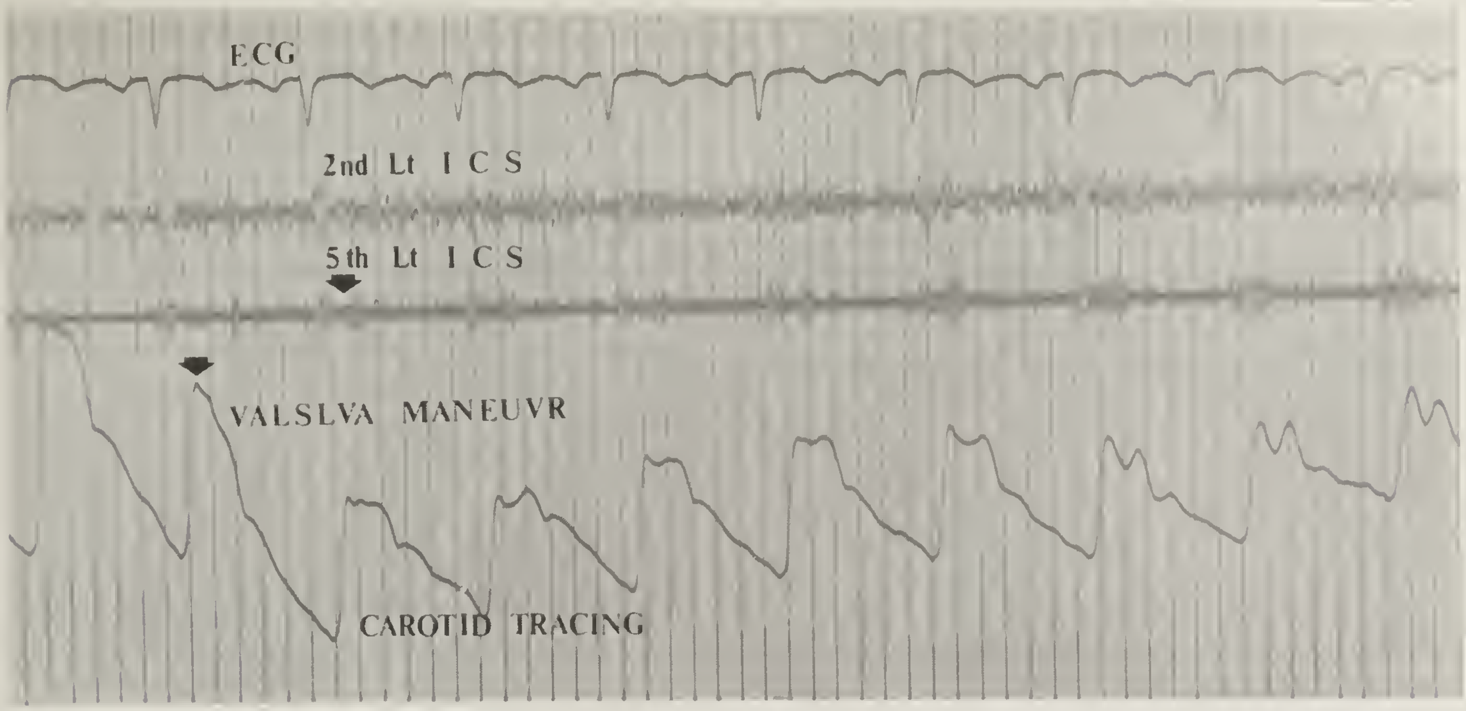

Figure 2

The effect of Valsalva maneuver on the simultaneously recorded ECG, phonocardiogram and carotid tracing. Note the steady decline in the amplitude of carotid impulse and increased intensity of the systolic murmur.

performed. In both studies there was a clear decrease in the amplitude of the carotid tracing concommitent with accentuation of the systolic murmur and the midsystolic dip of the carotid impulse. Prior to amyl nitrate inhalation his Pre-Ejection Phase/Left Ventricular Ejection Time (PEP/LVET) corrected for heart rate, was 63/263= 0.242 (Predicted 95/260= 0.365). Thirty seconds after amyl nitrate inhalation, the ratio changed to 52/280= 0.186 (Predicted 91/243= 0.374). On propranolol, 160mg/day, the ratio was 89/282= 0.316 (Predicted 105/301= 0.349).

Echocardiographic studies (Fig. 4) showed the left atrium to be moderately dilated and the left ventricular cavity diameters were normal. There was significant thickening of the ventricular septum with a septal to left ventricular free wall thickness ratio of 13/8= 1.62 (Upper limit of

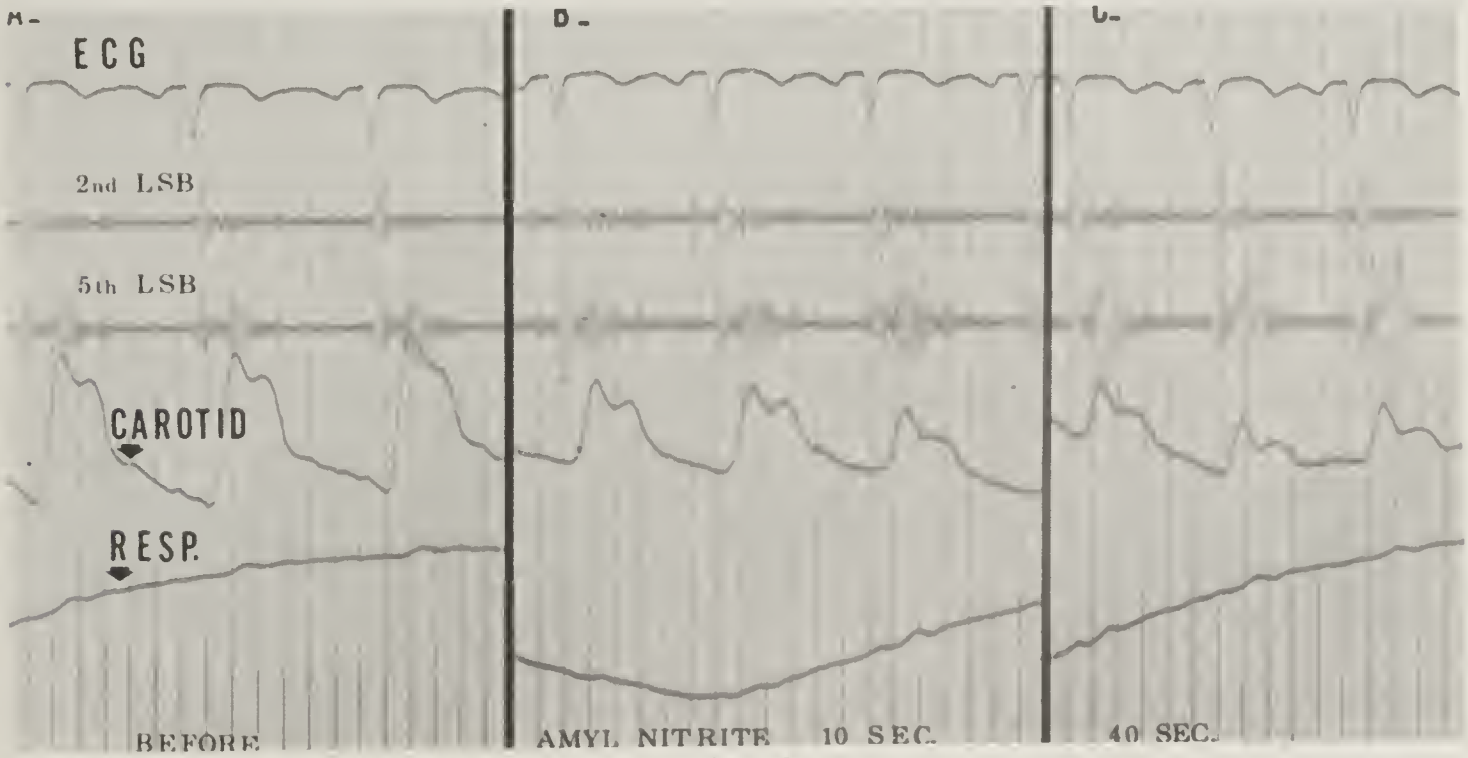

Figure 3

Simultaneous ECG, phonocardiogram, carotid tracing and pneumogram. A-Before amyl nitrate inhalation. B-Ten seconds, and C-40 seconds after amyl nitrate inhalation. Note, the increase in the intensity of the systolic murmur with decrease amplitude of the carotid impulse.

normal 1.3), and decreased systolic thickening. Decreased diastolic closure rate "E-F Slope" of the anterior leaflet of the mitral valve was noted. The leaflet came in contact with the left ventricular septum during ventricular diastole, with anterior motion towards the septum during systole. Clinical and non-invasive cardiac evaluations confirmed the diagnosis of idiopathic hypertrophic subaortic stenosis and cardiac catheterization was not considered indicated to further substantiate the diagnosis.

DISCUSSION

For over a decade cardiac catheterization and angiography were the only reliable methods for the diagnosis of IHSS. Even then, some cases remained undetected, unless a high degree of suspicion was present and adjunct provocative measures were employed during the catheterization procedures. In recent years, reliable echocardiographic criteria for the diagnosis of IHSS have been established.[3 4 5]

When echocardiographic examinations are performed in conjunction with apex and phonocardiography and carotid sphygmography, particularly with the use of provocative tests, such as Valsalva maneuver, amyl nitrite inhalation and vasoactive agents, even mild or moderate cases of IHSS can be detected. These tests may be repeated as frequently as required in studying the natural history of the disease, clinical course, and the response of the patients to various therapeutic measures. With the availability of non-invasive techniques, more cases of IHSS will be diagnosed which might otherwise have remained undetected, owing to atypical or minimal symptoms not severe enough to justify cardiac catheterization and angiography.

In spite of numerous publications relating to IHSS, the exact incidence of this disorder is not known. In recent years, several reports of bacterial endocarditis superimposed on IHSS have appeared in the literature, indicating a higher than expected incidence of bacterial endocarditis in such patients. Frank and Braunwald[6] reported 3 proven, one probable and 2 possible cases of bacterial endocarditis in 126 patients with IHSS with an incidence of 2.4 to 5%. Oakley[7] had seen 4 cases of bacterial endocarditis in 105 cases of IHSS and in 2 it proved fatal. Epstein et al.[8] reported 2 cases from a total of 22 and Wong et al.[9] 2 cases among 26 patients with IHSS. The overall incidence of bacterial endocarditis in these 4 groups is about 4-5%. The incidence of

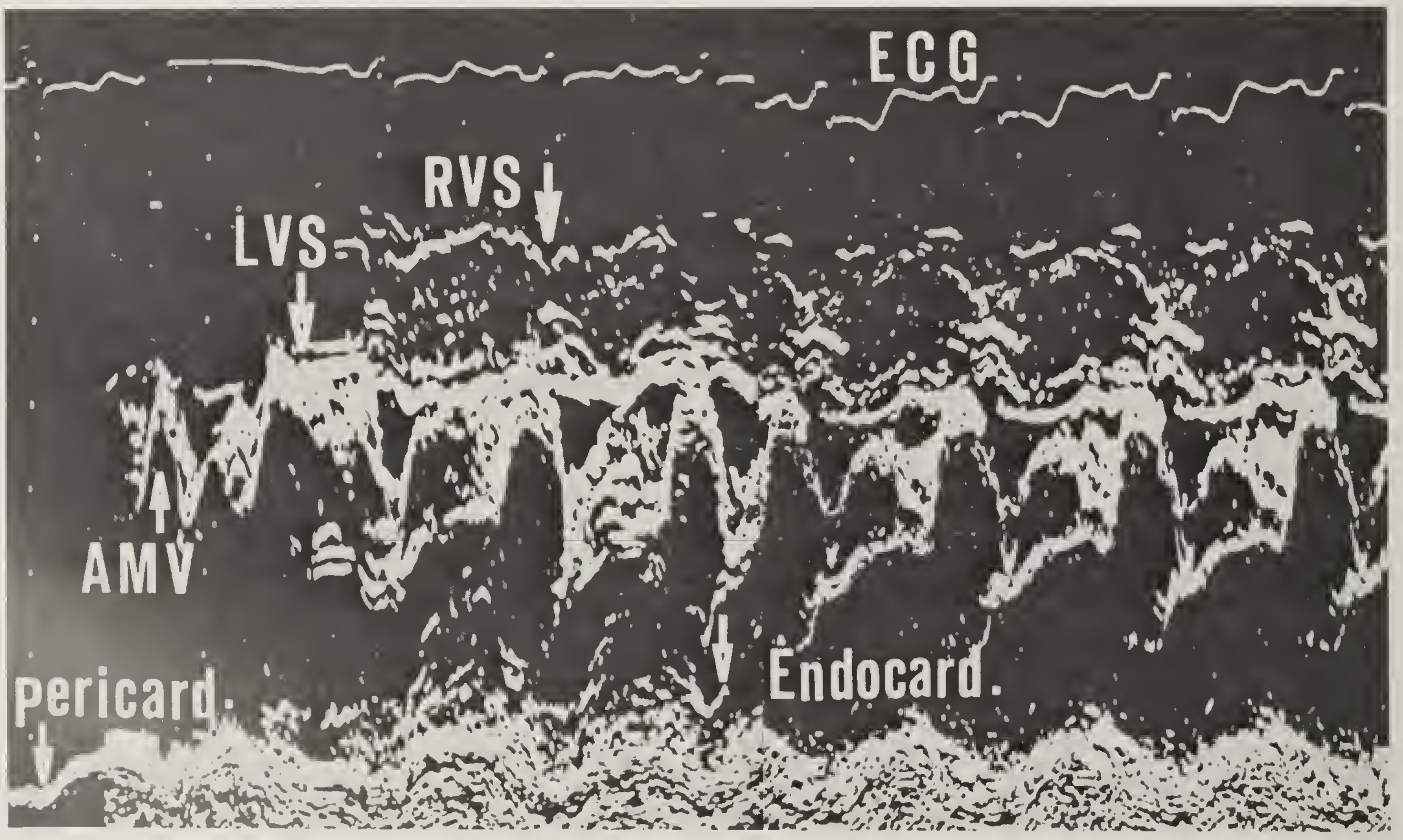

Figure 4
Echocradiogram reveals thickened ventricular septum (RVS-LVS) with reduced septal excursion. The septum is much thicker than the left ventricular free wall. The anterior leaflet of the mitral valve (AMV) comes in contact with the left ventricular septum during the diastole, with anterior displacement during systole. The diameter of the left ventricular cavity is normal.

bacterial endocarditis in the general population is about 2.4/100,000,[10] and 1/1,000 to 1/6,000 hospital admissions.[11] Cherubin[12] reported 0.54% of admissions in the period of 1939 - 1947, 0.40% from 1948 - 1957, and 0.32% from 1958 - 1967 due to endocarditis. In autopsy studies, the incidence of bacterial endocarditis was reported as 1.85% in the pre-antibiotic era in contrast to 0.47% in the antibiotic era.[13] The incidence of bacterial endocarditis in rheumatic heart disease has been reported to be between 5 to 10%, and 6.5% in all patients having congential heart disease.[14,15,16]

Summary review of the 16 proven cases of bacterial endocarditis superimposed on IHSS reported in the literature, are outlined in Table 1. Bacteriological studies identifying the infecting organisms were available in 14 cases. Most cases were caused by low virulence organisms known to cause subacute bacterial endocarditis in patients with underlying rheumatic valvular damage, congential cardiac defects and degenerative cardiac disorders. Eight cases (57%) were infected by Strep. Viridans or Alpha hemolytic Streptococcus. In 7 cases (50%), the endocarditis was considered to be subsequent to an oral, urogenital or catheterization procedure.

Pathogenesis of subacute bacterial endocarditis and pathophysiologic processes involved in its development have been well studied.[22,23] A previously damaged cardiac valve or a hemodynamic situation which produces pressure changes across an anatomic structure cause turbulance (jet effect), with the impinging high velocity blood flow injuring the surface by frequent and forceful impact a short distance beyond the anatomic alteration or constriction.[24] The pre-existing endothelial inflamation in association with the jet lesion, by presenting a sticky platelet-fibrin surface, favors the localization of blood borne bacteria on the low pressure side of the lesion, leading to endocardial infection. The process is further abetted by the high titer of agglutinin antibody for the infecting organism which is usually present in patients with subacute bacterial endocarditis.[23] This type of infection has been described as occurring, not only in congential defects and acquired valvular lesions at the site of surgical procedures and intracardiac prostheses, but also on intraventricular mural thrombi associated with myocardial infarction and in degenerative valvular disorders.[25,26]

Anatomic studies of the heart in IHSS have demonstrated marked increase in heart weight, involving mostly the left ventricle.[27] Severe asymmetrical septal hypertrophy with reduced or normal sized ventricular cavity and left atrial dilatation, fibrous thickening and degenerative changes of the mitral valve, particularly the anterior leaflet and chordae tendinea are frequently found. These may be associated with abnormalities in the length of the chordae.[28] Gross subendocardial scars and fibrosis of the papillary muscle have been described. Thickening and opacity of the ventricular endocardium may be present, which when severe and localized to the left ventricular outflow tract resembles discrete congenital subvalvular stenosis with secondary hypertropy.[29] These anatomical abnormalities are frequently associated with varying degrees of mitral regurgitation, which further predisposes the patient to bacterial endocarditis.

Okell[30] has demonstrated the development of

AUTHOR	AGE	SEX	INFECTING ORGANISM	VALVE INVOLVED	MODE OF DIAGNOSIS	PREDISPOSING PROCEDURE
Soulie', P., et al[2]	41	M	Strep. Viridans	-	Clinical	-
Frank, S., & Braunwald E.[6]	45	M	Strep. Viridans	Aortic	Surgery	Cardiac cath.
Frank, S., & Braunwald E.[6]	61	F	Strep. Viridans	-	Clinical	-
Frank, S., & Braunwald E.[6]	32	F	Not available	-	Clinical	-
Epstein, E. J., et al[8]	20	F	Strep. Fecalis	-	Clinical	Dental extraction
Epstein, E. J., et al[8]	39	M	Diphtroid Bacili	-	Clinical	Cardiac Cath.
Wang, K., et al[9]	43	M	Entrococci	Ao & Mit.	Autopsy	-
Boiteau, G. M., et al[17]	37	F	Strep. Viridans	-	Clinical	Dental extraction
Boiteau, G. M., et al[17]	23	F	Strep. Viridans	Mitral	Autopsy	Thraputic abortion
Linhart, J. W., et al[18]	29	M	Alpha H. Strep.	-	Catheter	Dental extraction
Nagle, J. P.[19]	34	M	Staph. Epidemis	Aortic	Autopsy	-
Vecht, R. J., et al[20]	39	M	On antibiotic	Mitral	Autopsy	Dental filling
Vecht, R. J., et al[20]	49	F	Peumococcal	-	Expired	-
Vecht, R. J., et al[20]	59	F	Staph. Albus	-	Clinical	-
Gardelia, J. U., et al[21]	58	M	Strep. Viridans	Aortic	Catheter	-
Present study	54	M	Alpha H. Strep.	-	Clinical	-

Table 1

Cases with Idiopathic Hypertrophic Subaortic Stenosis and infective endocarditis reported in the literature transient bacteremia proven by positive blood cultures in up to 72% of patients who have undergone oral or dental procedures. Instrumentation of the urinary tract initiated transient bacteremia in 10 to 40% of patients. Hobbson and Juel[31] found that Strep. Viridans endocarditis occurred in a significant proportion of persons from whom teeth had been extracted within the previous 42 days. This is consistent with the development of bacterial endocarditis following oral, urogenital and catheterization procedures in 50% of reported cases of bacterial endocarditis associated with IHSS. The majority of the cases were caused, not by infectious processes or highly invasive micro-organisms, but by organisms which normally reside as components of the indigenous human flora, initiating endocarditis only if there is an underlying cardiovascular lesion.

Thus, the reported 5% incidence of bacterial endocarditis associated with IHSS in the presence of anatomic cardiac lesions and hemodynamic derangement, especially mitral regurgitation, indicate similar pathogenetic mechanisms which come into play in the endocardial infections of IHSS and those of rheumatic valvular or congential heart disease. In approximately half of the reported cases of endocarditis occurring in patients with IHSS, the infection developed following some type of dental or other surgical procedure, indicating the high degree of susceptibility of these patients to bacterial seeding. Individuals with IHSS should not only receive adequate antibiotic prophylaxis prior to dental or other predisposing procedures, but the importance of reporting febrile illnesses should be emphasized as well.

SUMMARY

Idiopathic hypertrophic subaortic stenosis is a rare disease. The diagnosis is usually suspected clinically, and proven by catheterization procedures or non-invasive studies.

The incidence of bacterial endocarditis associated with IHSS is about 5%. The pathogenesis and pathophysiologic mechanism of bacterial endocarditis in IHSS appear similar to those in rheumatic and congenital heart disease. History of a prior predisposing procedure in 50% of cases of B. E. and IHSS indicates the high degree of susceptibility of these patients to the infection and emphasizes the need for prophylactic antibiotic administration to the patients prior to such procedures.

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THE OMINOUS CLEAR LUNG OF COPD (CHRONIC OBSTRUCTIVE LUNG DISEASE)

SAMUEL L. COHEN, M.D.

INTRODUCTORY SUMMARY

The author presents examples of patients with severe chronic obstructive pulmonary disease in whom marked aggravation of the clinical condition was accompanied by an increase in the degree of pulmonary hyperlucency. The sequence of events is compared to obstructive bronchiolitis in infants in whom apparent improvement in aeration, as seen on the roentgenogram, might actually be accompanied by an aggravation of the clinical condition.

ABSTRACT

The apparent increase of chronic obstructive lung disease and the newer methods of treatment have brought to our attention a paradoxical radiographic finding that must be recognized and understood to assure proper management of the patient in respiratory failure. The finding of "clear" lungs on serial radiographs may indicate a worsening of the patient's pulmonary compensation rather than an improvement in his condition.

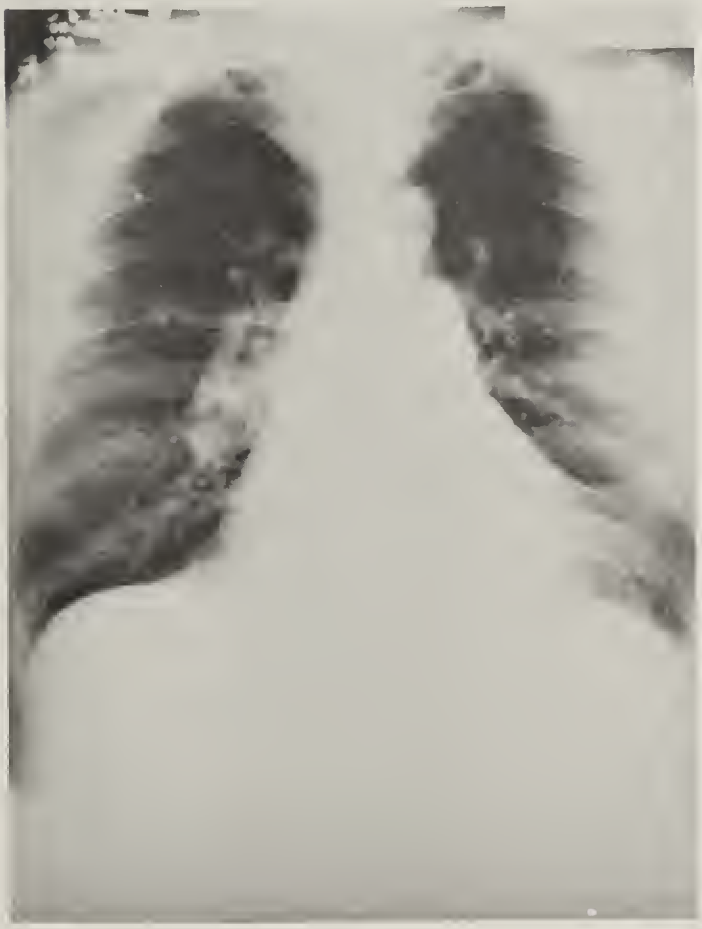

Figure 1a

Case 1a (P.Z.) Chronic severe COPD

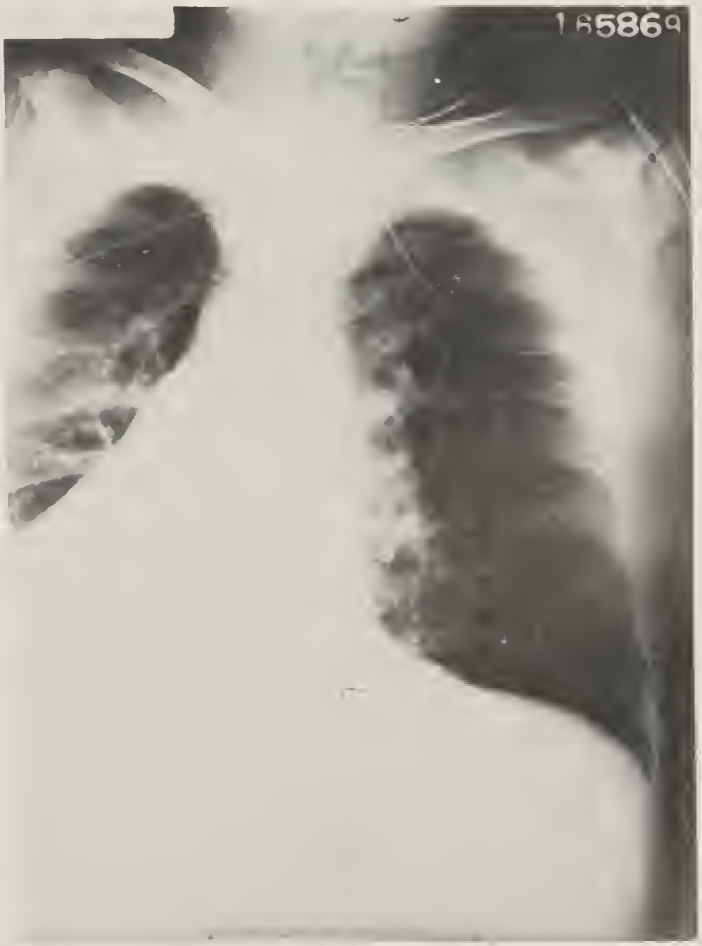

Figure 1b

Case 1b (P.Z.) In extremis with abnormal blood gases with hyperexpanded "clear lungs".

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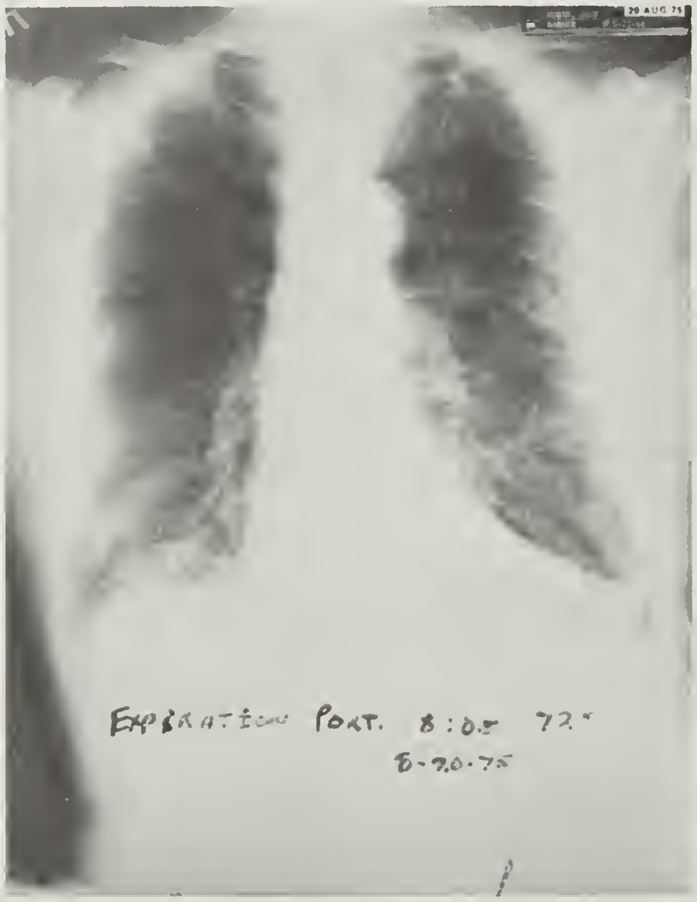

Figure 2a

Case 2a (J.R.) Chronic severe COPD.

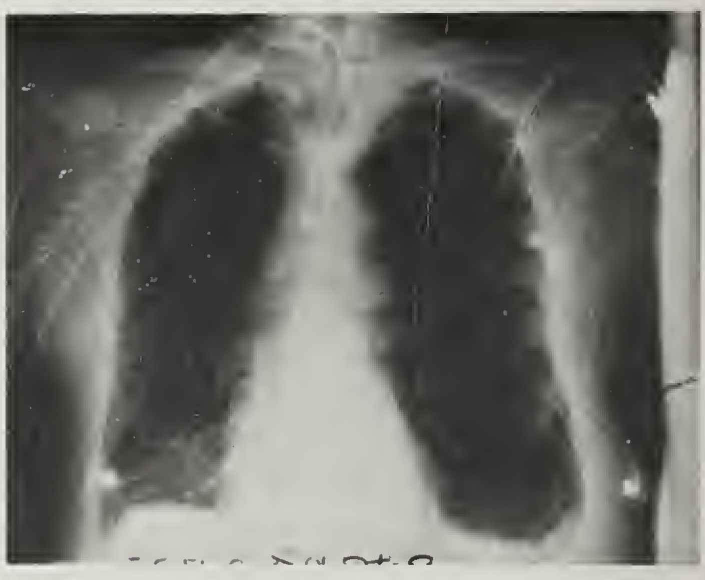

Figure 2b

Case 2b (J.R.) In extremis with "clear lungs" hyperexpanded with abnormal blood gases.

It is well recognized that in infant peripheral bronchiolitis the chest x-rays may show no parenchymal infiltrates, but will indicate "air trapping" and not much else.[2]

It is also well recognized that chronic obstructive lung disease is associated with hyperexpanded lungs.[2] In the course of interpreting a large number of chest roentgenograms in Phoenix in private practice and at the Phoenix Veterans Hospital throughout the past 20 years, an extraordinary number of patients with COPD were seen. It became evident that in a number of cases, when these patients were under pulmonary intensive care and extremely ill,[3] the chest roentgenogram would suddenly clear and the lungs would be hyperexpanded. From this one would think that the patient was doing quite well. However, when the patients were examined by clinicians, it was evident that they were in extremis and when laboratory studies were done, blood gases, etc., the results would be quite abnormal.

It, therefore, in our opinion, is of interest and perhaps of value, to report two cases. It is felt that when these findings are present, it behooves the radiologist not only to report that the lungs are more clear but to suggest that this might represent bronchiole or bronchial obstruction and that there is a possible poor exchange of gases through the lung membranes.

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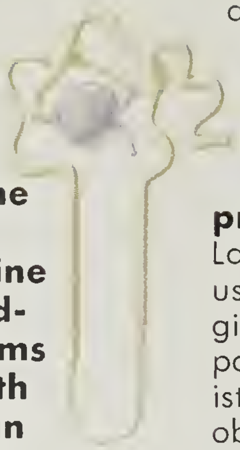

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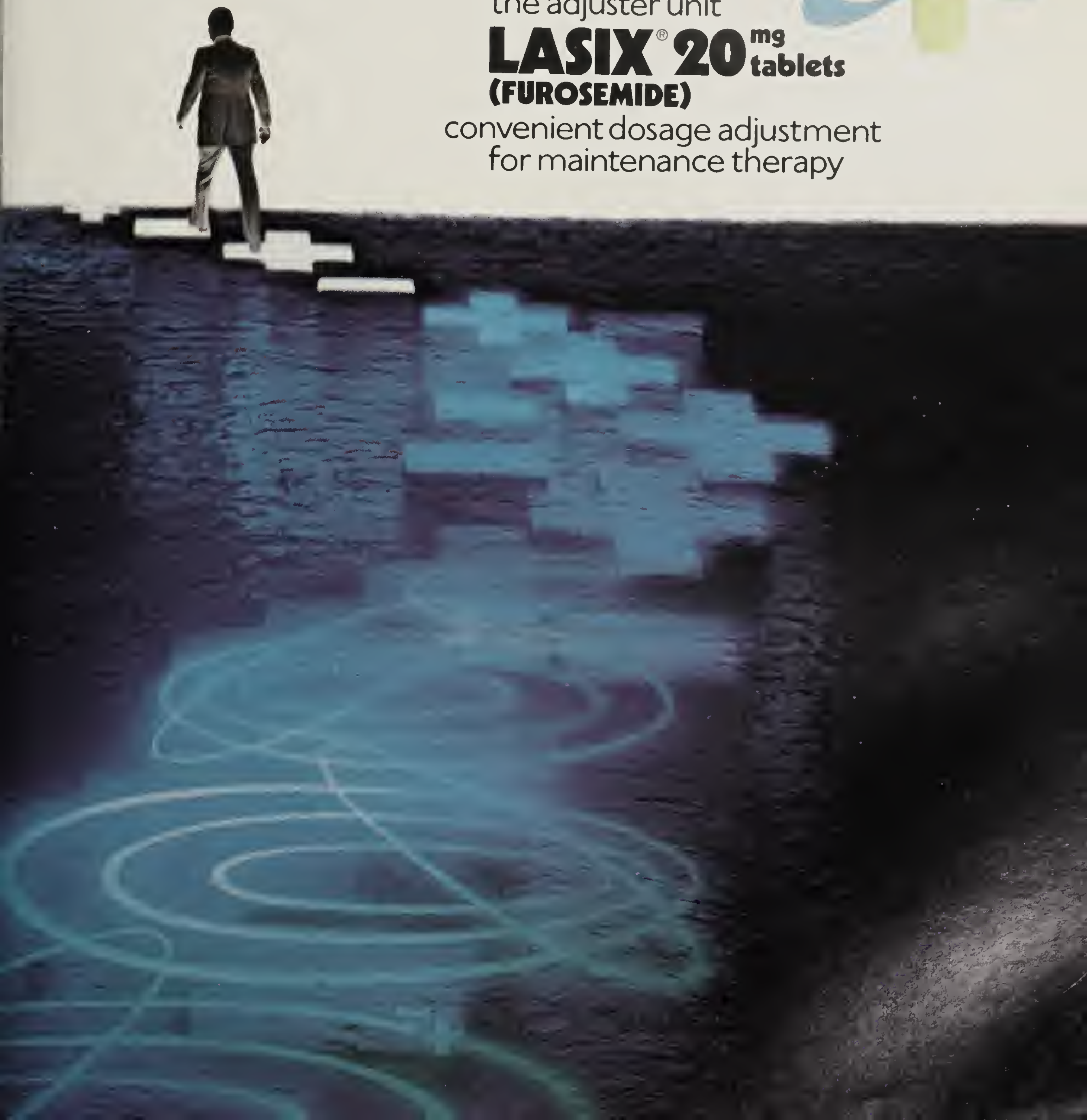

LASIX® 40 mg and 20 mg tablets. (FUROSEMIDE)

logical long-term diuretic therapy for cardiac edema

A brief summary of the Prescribing Information for

Lasix® (furosemide) Tablets 20 mg and 40 mg

WARNING—Lasix (furosemide) is a potent diuretic which if given in excessive amounts can lead to a profound diuresis with water and electrolyte depletion. Therefore, careful medical supervision is required, and dose and dose schedule have to be adjusted to the individual patient's needs. (See under "Dosage and Administration.")

Indications—Lasix (furosemide) is indicated for the treatment of the edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Hypertension—Lasix (furosemide) may be used for the treatment of hypertension alone or in combination with other antihypertensive drugs. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controllable with Lasix (furosemide) alone.

CONTRAINDICATIONS—Because animal reproductive studies have shown that Lasix (furosemide) may cause fetal abnormalities, the drug is contraindicated in women of childbearing potential. (See "Additional Information.")

Lasix (furosemide) is contraindicated in anuria. If increasing azotemia and oliguria occur during treatment of severe progressive renal disease, the drug should be discontinued. In hepatic coma and in states of electrolyte depletion, therapy should not be instituted until the basic condition is improved or corrected. Lasix (furosemide) is contraindicated in patients with a history of hypersensitivity to this compound.

Warnings—Excessive diuresis may result in dehydration and reduction in blood volume, with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients. Excessive loss of potassium in patients receiving digitalis glycosides may precipitate digitalis toxicity. Care should also be exercised in patients receiving potassium depleting steroids.

Frequent serum electrolyte, CO_2 and BUN determinations should be performed during the first few months of therapy and periodically thereafter, and abnormalities corrected or the drug temporarily withdrawn.

In patients with hepatic cirrhosis and ascites, initiation of therapy with Lasix (furosemide) is best carried out in the hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma; therefore, strict observation is necessary during the period of diuresis. Supplemental potassium chloride and, if required, an aldosterone antagonist are helpful in preventing hypokalemia and metabolic alkalosis.

Patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

In those instances where potassium supplementation is required, an oral liquid preparation should be used rather than enteric-coated potassium salts.

There have been several reports, published and unpublished, concerning nonspecific small-bowel lesions consisting of stenosis, with or without ulceration, associated with the administration of enteric-coated thiazides with potassium salts. These lesions may occur with enteric-coated potassium tablets alone or when they are used with nonenteric-coated thiazides, or certain other oral diuretics.

These small-bowel lesions have caused obstruction, hemorrhage, and perforation. Surgery was frequently required, and deaths have occurred.

Available information tends to implicate enteric-coated potassium salts, although lesions of this type also occur spontaneously. Therefore, coated potassium-containing formulations should be administered only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Patients with known sulfonamide sensitivity may show allergic reactions to Lasix (furosemide).

Precautions—As with any potent diuretic, electrolyte depletion may occur during therapy with Lasix (furosemide), especially in patients receiving higher doses and a restricted salt intake. Electrolyte depletion may manifest itself by weakness, dizziness, lethargy, leg cramps, anorexia, vomiting, and/or mental confusion.

Asymptomatic hyperuricemia can occur and gout may rarely be precipitated. Reversible elevations of BUN may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

When parenteral use of Lasix (furosemide) precedes its oral use, it should be kept in mind that cases of tinnitus and reversible hearing impairment have been reported. There have also been some reports of cases in which irreversible hearing impairment occurred. Usually, ototoxicity has been reported when Lasix (furosemide) was injected rapidly in patients with severe impairment of renal function at doses exceeding several times the usual recommended dose and in whom other drugs known to be ototoxic were often given. If the physician elects to use high dose parenteral therapy in patients with severely impaired renal function, controlled intravenous infusion is advisable (for adults, it has been reported that an infusion rate not exceeding 4 mg Lasix [furosemide] per minute has been used).

Increases in blood glucose, and alterations in glucose tolerance tests with abnormalities of the fasting and two-hour postprandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Lasix (furosemide) may lower serum calcium levels, and rare cases of tetany have been reported.

Patients receiving high doses of salicylates, in conjunction with Lasix (furosemide) may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Diuretics such as furosemide may enhance the nephrotoxicity of cephaloridine. Therefore, Lasix (furosemide) and cephaloridine should not be administered simultaneously.

Sulfonamide diuretics have been reported to decrease arterial responsiveness to pressor amines and to enhance the effect of tubocurarine. Great caution should be exercised in administering curare or its derivatives to patients undergoing therapy with Lasix (furosemide), and it is advisable to discontinue Lasix (furosemide) for one week prior to any elective surgery.

Adverse Reactions—Various forms of dermatitis, including urticaria and rare forms of exfoliative dermatitis, erythema multiforme, pruritus, paresthesia, blurring of vision, postural hypotension, nausea, vomiting, or diarrhea.

Anemia, leukopenia, aplastic anemia, and thrombocytopenia (with purpura). Rare cases of agranulocytosis which responded to treatment.

In addition, the following rare adverse reactions have been reported; however, relationship to the drug has not been established with certainty: sweet taste, oral and gastric burning, paradoxical swelling, headache, jaundice, thrombophlebitis and emboli and acute pancreatitis.

Lasix (furosemide)-induced diuresis may be accompanied by weakness, fatigue, lightheadedness or dizziness, muscle cramps, thirst, increased perspiration, urinary bladder spasm, and symptoms of urinary frequency.

Dosage and Administration

ADULTS

The usual adult dose of Lasix (furosemide) is 20 to 80 mg given as a single dose.

If the diuretic response with a single dose of 20 to 80 mg is not satisfactory, the following schedule should be used: Increase this dose by increments of 20 or 40 mg not sooner than 6 to 8 hours after the previous dose until the desired diuretic effect has been obtained. This individually determined single dose should then be given once or twice daily. The dose of Lasix (furosemide) may be carefully titrated up to 600 mg per day in those patients with severe clinical edematous states.

With doses exceeding 80 mg/day and given for prolonged periods, careful clinical and laboratory observations are particularly advisable.

Hypertension—The usual dose of Lasix (furosemide) is 40 mg twice daily both for initiation of therapy and for maintenance. Careful observations for changes in blood pressure must be made when this compound is used with other antihypertensive drugs, especially during initial therapy. The dosage of other agents must be reduced by at least 50 percent as soon as Lasix (furosemide) is added to the regimen to prevent excessive drop in blood pressure. As the blood pressure falls under the potentiating effect of Lasix (furosemide), a further reduction in dosage, or even discontinuation, of other antihypertensive drugs may be necessary. It is further recommended, if 40 mg twice daily does not lead to a clinically satisfactory response, to add other hypotensive agents, e.g., reserpine, rather than to increase the dose of Lasix (furosemide).

INFANTS AND CHILDREN

Pediatric Administration: The usual initial dose of oral Lasix in infants and children is 2 mg/kg body weight, given as a single dose. If the diuretic response is not satisfactory after the initial dose, dosage may be increased by 1 or 2 mg/kg not sooner than 6 to 8 hours after the previous dose. Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

How Supplied—Lasix Tablets 40 mg (furosemide) supplied as white, round, monogrammed, scored tablets.

Lasix Tablets 20 mg (furosemide) supplied as white, oval, monogrammed tablets.

Note: Dispense in dark containers. Exposure to light may cause slight discoloration which, however, does not alter potency.

Additional Information

Toxicology

The acute toxicity of Lasix (furosemide) has been determined in mice, rats, and dogs. In all three animal species, the oral LD_{50} of Lasix (furosemide) exceeded 1000 mg/kg of body weight, while the intravenous LD_{50} ranged from 300 to 680 mg/kg. Intragastric injection of the drug in newborn rats resulted in an LD_{50} of 380 mg/kg.

The acute toxicity of high doses of Lasix (furosemide) was characterized by convulsions, paralysis, and collapse. Surviving animals often became dehydrated and depleted of electrolytes due to the diuresis induced by Lasix (furosemide). In the newborn rats, intragastric injection of the drug caused hyperactivity and anorexia.

Chronic toxicity studies with Lasix (furosemide) were done in rats and dogs. In a one-year study in rats, renal tubular degeneration occurred, with all doses higher than 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day). A six-month study in dogs revealed calcification and scarring of the renal parenchyma at all doses above 10 mg/kg (83 percent of the maximal recommended human dose of 600 mg per day).

Reproductive Studies

The effects of Lasix (furosemide) on embryonic and fetal development and on pregnant dams were studied in mice, rats, and rabbits.

Lasix (furosemide) caused unexplained maternal deaths and abortions in the rabbit when 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day) was administered between days 12 to 17 of gestation. In a previous study the lowest dose of only 25 mg/kg (2 times the maximal recommended human dose of 600 mg per day) caused maternal deaths and abortions. In a third study, none of the pregnant rabbits survived a dose of 100 mg/kg. Data from the above studies indicate fetal lethality which can precede maternal deaths.

The results of the mouse study and one of the three rabbit studies also showed an increased incidence of hydronephrosis (distention of the renal pelvis and, in some cases, of the ureters) in fetuses derived from treated dams as compared to the incidence in fetuses from the control group.

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PROFESSIONAL COURTESY — A DYING AMENITY

JOHN W. KENNEDY, M.D.

Professional courtesy, like other customs and cultural mores, is changing. Or perhaps it is only the unusual incidents that catch our attention, and the basic custom is unaltered.

Not too long ago it was unthinkable to tender a professional fee to a physician or his immediate family. It was considered an honor and a privilege to be selected by a colleague as his medical advisor. Under present economic circumstances this had to change and most of us feel that it changed for the better.

WW II marked the end of that era of professional courtesy. It was not uncommon for physicians in service—whose family had been uprooted to a strange city or village, near an Army or Navy training area, to find that the local physician rendered and expected a fee for his services.

Thereafter, many of us began to carry health insurance, since many sensed the inequity of taking advantage of our fellow physicians' professional courtesy. It was hoped that the performing physician would accept this insurance fee as at least a partial compensation for his time and skill, and goodness knows, I have received generous attention and skill, in this very valley, from many of my fellow physicians.

But when this change in professional courtesy custom disappeared, some professional empathy, or so it seems so to me as the cerebral cortex shinks away from the calvarium, perhaps it only seems that way. So what are we concerned about —that professional empathy for the sick and dying physician may be just as lacking as our alleged lack of empathy to patients in general. Every newspaper and periodical reminds us of this circumstance.

A case in point is a retired physician, here in the valley. He had been feeling ill for some time, but suddenly became acutely ill, with fever and chest pain. The wife called her medical advisor, asking him if he couldn't please stop by and see her physician husband to see if it was necessary to hospitalize him, or what she should do. A preemptory, "No, I cannot make a house call. No, no one else in my office does." (that is the generally accepted new tradition) "I know of no one else who does, and you should recognize that your husband is dying and take him to a rest home". The advice may have been proper, but the empathy was totally lacking.

Since we must all eventually travel this lonely road, let's keep some empathy available in the bank, indeed most of the profession does carefully renew and treasure it, to some it appears too burdensome.

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President's Page

A PARADOX OF PRESSURES

EDWARD SATTENSPIEL, M.D.

There is a paradox of pressures on the medical profession today. From one side, we are exhorted about our responsibilities to provide first class medical care for everyone. On the other side, there are constant attacks from various groups of "health professionals" trying to expand the scope of their activities beyond their defined roles and capabilities, thereby encroaching more and more on the traditional practice of medicine with what can only be called second class care.

Every year the legislature is besieged with bills along these lines . . . Proposals to require hospitals to grant major surgical in-patient privileges to podiatrists; to force insurance carriers to pay for chiropractic care, psychology services, etc., to license various types of medical technicians; to permit optometrists to use drugs; and on and on and on.

Let's look at the Optometry problem for a minute. They almost succeeded in getting their bill, which would allow them to use drugs in the eye, through the last session of the legislature. Truman Plainer, M.D., president of the Arizona Ophthalmological Society, has clearly stated that it is our duty to alert the public to the dangers inherent in the proposed law. It is only through the strong and continuing efforts of the Ophthalmologists to properly inform our legislators that this bill has so far been blocked. Optometry is a measuring science concerned with the refractive powers of the human eye. It is not a medical science and its practitioners are not sufficiently trained to safely use drugs or accurately diagnose disease. Yet, the pressure of the Optometry lobby is enormous. In their state journal, a director of their state society has attacked all Ophthalmologists who have testified against their bill, labelling them as "disastrous, professionally and politically" and he goes on to list the names of eleven such Ophthalmologists in Arizona (including the chairman of Ophthalmology at the College of Medicine, and the president of the state Ophthalmological Society). He asks all optometrists to join in economic sanctions against these physicians!

We, as physicians, are not concerned with the economic implications raised by those who are trying to erode the practice of medicine. We are concerned about all those efforts which jeopardize the standards of health care and open the door to truly second class, inferior medical care. All physicians must join in the continuing battle to preserve our high standards against attack from any quarter.

There are influential legislators who seek the opinions of the Arizona Medical Association and individual M.D.'s in all these areas. Accordingly, the primary charge of our professional committee this year is to review all legislative material, past and present, in these controversial areas and develop position papers reflecting an objective balanced approach to the maintenance of high standards of health care for the people of Arizona, to be presented to the legislature whenever needed.

Dean's Page

HUMAN BEHAVIOR AND DEVELOPMENT

NEAL A. VANSELOW, M.D.

Doctor, should I get a divorce? . . . Doctor, my child has cerebral palsy and we want a healthy baby, should I get pregnant again? . . . Doctor, my mother is old and helpless, should I put her in a nursing home? . . . Doctor, I think my 14-year old is on drugs, what should I do? . . . Doctor, my husband is an alcoholic, what shall I do? . . . Doctor, our sex life is terrible, will you help us?

These, and many other kinds of inquiries, are typical problems that daily confront and confound physicians in practice. Four years ago, a new required course was instituted at the College of Medicine which includes discussion of issues like those above, as well as many other aspects of the physician-patient-family relationship, ethical issues in medicine, and normal growth and development patterns throughout the life span. Basically, the goals of the course, called Human Behavior and Development, are to provide today's medical students with information and understanding about the psychological, social and cultural factors that interact with a patient's health or illness to bring that patient to a physician for help. Although obviously related to what students are learning in their other basic science courses and what they will learn later in their clinical training, the focus of this course is on aspects of medical practice which are not treated systematically elsewhere.

The course follows a developmental sequence with the periodic insertion of topics which are not specifically age-related. Starting with the prenatal period and following through the lifespan to death and dying, the emphasis is on the dynamics of human behavior as it relates to the physician and the patient. Initial sessions deal with genetic counseling, and how the physician can provide the support and information parents need to make decisions about having a baby. A session on the malformed baby examines how the physician informs the parents of the baby's problems, the resources that are available, and the kinds of support the parents need from the physician and others. The course of normal growth and development from birth through childhood is reviewed, as well as some of the significant factors in psycho-social development of the child. The increasingly common situation of having both parents employed, and the advantages and disadvantages of day care for children is considered. The sensitivity of the physicians' role in instances of sudden infant death and child abuse is also considered.

Later, issues in dealing with adolescent patients are discussed, as well as a number of issues relating to the principal reproductive years—marriage and divorce, conception, sterility, birth control, childbirth, venereal disease, abortion, and rape. Other sessions are devoted to understanding human sexuality, and the physician's role in helping patients with sexual problems. Alcoholism and substance abuse are discussed in sessions which focus on how the physician can be most helpful both to the abuser and affected family members.

Toward the end of the course, the management of patients with chronic illness is discussed, the problems of aging, and the difficulties of coping with death and dying—for the physician, the patient and the patient's family. Some of the major legal issues involved in the practice of medicine are discussed, as well as numerous ethical issues which arise in practice—for example, the issues involved in organ transplants, euthanasia, abortion, informed consent, and the differing religious and moral views of the physician and the patient.

In one sense, the whole course is directed toward providing some guidelines and answers to the question, "What is a good doctor?" The first class session poses this question and the rest of the course provides a variety of approaches and answers to the question. The course has two coordinators, one a physician in private practice and one a social psychologist, who themselves teach a number of the class sessions and also invite experts in many different areas to present ideas and information from their own fields. The emphasis in all class sessions is on interaction between faculty and students, and traditional lectures are kept to a minimum. Patient interviews, panel discussions, small group discussions and simulated role-playing situations are used to present what are often very complex topics with no easy answers. The two coordinators are present at all class sessions to help keep the sessions focused and to provide continuity for the course as a whole.

The response to the course by students over the last four years has been very positive. One measure of this is the number of students who have contacted the coordinators during the remainder of their medical school training for additional help with specific problems which have arisen when they are caring for patients. Students have affirmed the importance of the course in preparing them to deal more effectively with patients through evaluation of the sessions while the course is in progress, as well as evaluations two years later after they have completed their clinical training.

Nationally, our students have done outstandingly well in the portion of the National Board examination which covers the material presented in the course. In contrast to the years prior to the introduction of the course, the mean score for students at our medical school since the course was instituted has consistently placed us among the top ten schools in the country on this portion of the National Board examination.

Neal A. Vanselow

Clinical Oncology
In Arizona

THE PRELEUKEMIC SYNDROME

TERENCE S. HERMAN, M. D.
STEPHEN E. JONES, M. D.

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Physicians not infrequently encounter patients who present with unexplained cytopenias that are refractory to hematinic therapy, and it has been recognized for many years that a portion of these patients will either eventually develop acute leukemia or die from profound depression of peripheral blood counts. Because there have been no pathognomonic laboratory or clinical findings, this *"preleukemic syndrome"* has remained the least well defined of the genetic and acquired disorders known to be associated with the eventual emergence of leukemia (see Table I). Evaluation of these patients has served to rule out the few correctable causes of the hematologic abnormalities, but it has not been possible to predict which patients would follow a benign or malignant course. Recently, however, there have been important advances in laboratory methods which may provide a clearer definition of the preleukemic syndrome and help the clinician in assessing the prognosis of individual patients. This paper will review the clinical and laboratory features of the preleukemic syndrome with special emphasis upon these new laboratory techniques.

CLINICAL AND HEMATOLOGIC FINDINGS IN THE PRELEUKEMIC SYNDROME

The preleukemic syndrome occurs most commonly in men and about 70% of patients are over the age of 50. The evolving leukemia usually is in either the stem cell, myelocytic, or myelo-

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TABLE I
CONDITIONS ASSOCIATED WITH THE EVENTUAL DEVELOPMENT OF ACUTE LEUKEMIA
(A Partial List)*

- Chronic granulocytic leukemia
- Agnogenic myeloid metaplasia
- Polycythemia rubra vera
- Fanconi's anemia (hypoplastic anemia with multiple congenital abnormalities and chromosomal aberrations)
- Bloom's syndrome (infants with low birth weight, stunted growth, telangiectatic skin disorder, and chromosomal abnormalities)
- Sex chromosome abnormalities (Klinefelter's syndrome, mosaicism)
- Mongolism
- Ataxia telangiectasia
- Congenital agranulocytosis
- Bone marrow injury from
    - -Radiation
    - -Drugs or chemicals (benzene, phenylbutazones, chloramphenicol, cancer chemotherapeutic agents)
- "Preleukemic syndrome" (*see* Table II)

*After R. Pierre.[8]

monocytic category. (A review of an extensive literature reveals only two case reports of the development of lymphoblastic leukemia after preleukemic states.[1,2]) Although the incidence of the preleukemic syndrome is not known, reports of hematologic dysfunction predating acute leukemia vary from zero to about 30% of cases, and one author has estimated that a hematologic illness compatible with the preleukemic syndrome is probably present in as many as two of every five patients with acute leukemia.[3] Since the illness is often asymptomatic, it is obvious that many patients with the preleukemic syndrome may be missed.

Nonspecific symptoms usually referable to anemia are seen in about one half of identified cases of preleukemia. Bleeding or infection are rare as presenting symptoms. Physical findings are also nonspecific, but splenomegaly is present in about 20% of patients.[3]

Examination of the peripheral blood smear usually reveals a combination of cytopenias, with anemia being present in nearly all cases. Oval macrocytosis like that in folate or B^{12} deficiency is a frequent finding, but hypochromic microcytic anemia has also been recorded. A thorough search of the smear frequently reveals a few blasts or nucleated red cells. An absolute increase in monocytes is found in 10%-25% of patients and an increased number of bilobed granulocytes (Pelger-Heut anomaly) has been emphasized as a diagnostic point. Thrombocytopenia is present in about two thirds of patients, but it is rare as an isolated finding.[4]

Examination of the bone marrow specimen usually reveals qualitative and quantitative abnormalities of all cell lines. Hypercellularity is most common, but hypocellularity or normocellularity are not infrequent. Cells in the erythrocytic series are usually increased in number, with prominent nuclear immaturity for the degree of cytoplasmic differentiation as in B^{12} or folate deficiency.[3] Stainable iron is usually increased and is often present within ring sideroblasts (normoblasts which contain iron, a ring surrounding the nucleus). This is thought to reflect an inability of the bone marrow to utilize storage iron normally. Cells in the myeloid series are often increased in number, with a shift within this compartment to a predominance of early forms. An increase in promonocytes in the bone marrow is also a common finding. Megakarocytes may be increased in number and are often bizarre in shape.[5]

Other laboratory tests in the preleukemic syndrome are frequently abnormal. Leukocyte alkaline phosphatase scores (a measure of the lysosomal enzymes within neutrophils) are often low, and levels of plasma and urine muramidase (a breakdown product of lysosomal enzymes) are elevated in about 20% of cases, reflecting an increased turnover of myelocytes and monocytes. The folate and B_{12} levels are normal and the Coombs' test is negative. An unexplained mild increase in fetal hemoglobin is often present.[6] Increased susceptibility of red cells to complement-mediated lysis, like that seen in paroxysmal nocturnal hemoglobinuria, has been found in some patients.[7] A comparison of the typical findings of patients with acute leukemia, "smoldering" acute leukemia, and preleukemia is presented in Table II.

CHROMOSOMAL ANALYSIS AND BONE MARROW CULTURE

In the past few years, chromosomal analysis and bone marrow culture have been applied to the study of leukemic and preleukemic states. In one recent study conducted by Robert Pierre and colleagues at the Mayo Clinic,[8,9] it was found that 52% of overtly leukemic patients and about 27% of preleukemic patients had chromosomal abnormalities. In the preleukemic group, about 25% of patients with abnormal chromosomes compared to only about 8% of those with normal chromosomes have progressed to overt leukemia. When the preleukemic group was analyzed for survival, it was found that the preleukemic patients with chromosomal abnormalities had a strikingly increased mortality which could not be totally ascribed to the development of overt

TABLE II
CLINICAL AND HEMATOLOGIC FINDINGS IN PRELEUKEMIA, "SMOLDERING LEUKEMIA,"* AND ACUTE LEUKEMIA

	Preleukemia	"Smouldering" Leukemia*	Acute Leukemia
Peripheral Blood			
Anemia	+	+	+
↑ WBC	−	-	++ to ++++
↓ Platelets	+	+	++
Blasts	+	+	+++
Bone Marrow			
↑ Cellularity	±	+	+++
↑ Blasts	-	+ or ++	+++
Auer Rods	-	±	±
↓ Megakaryocytes	-	+	++
↓ RBC Precursors	-	+	++
Symptoms			
Fatigue	+	+	++
Purpura	(rarely +)	+	++
Infection	(rarely +)	+	++

*"Smouldering" leukemia is a syndrome of acute leukemia, gererally in older patients, which is slowly progressive (low growth fraction) and may be managed conservatively for a period of time (usually weeks to months) before intensive antileukemic therapy is required.

leukemia. This latter finding illustrates the fact that *the cytopenias of the preleukemic syndrome may be fatal in themselves even without the development of acute leukemia*, and that this may be especially true in preleukemics with chromosomal abnormalities. These findings are quite analogous to the reported data indicating that patients with overt leukemia who have chromosomal abnormalities also have significantly shorter survival as a group than do patients without chromosomal abnormalities.[10] (For a comprehensive review of specific chromosomal findings in patients with various hematologic diseases, *see* Rowley.[11])

The development of the soft agar bone marrow culture technique for the culture of colonies of leukocytes from heparinized bone marrow provides a method for assessing granulocytic proliferation and differentiation. The bone marrow cells will grow and differentiate only if a "feeder" layer of peripheral blood leukocytes is present beneath the surface layer of the agar upon which the heparinized bone marrow cells are placed. The peripheral blood leukocytes produce substances which diffuse through the intervening agar and stimulate the white cell colonies. In this system, the colony-stimulating activity (CSA) of the peripheral leukocytes can then be quantitated, and the pattern and differentiation of the white cell colonies observed.

Greenberg et al[12] studied the ability of normal, leukemic, and preleukemic bone marrows to produce white cell colonies. In contrast to normal marrow, marrow from patients with overt active leukemia produced either no colonies or excessive numbers of small, abortive colonies when cultured with peripheral leukocytes from normal volunteers as the source of CSA. Remission bone marrows produced normal colonies and abnormal colony formation was a forerunner of relapse. Marrow from patients with preleukemia produced almost no colonies in response to normal CSA factor. When the neutrophils of patients with overt active leukemia were used in the feeder layer for bone marrow from normal volunteers, it was found that CSA was less than 10% of normal. In contrast, the CSA for neutrophils from remission patients was normal or near normal. The CSA in preleukemics fell between the levels found in overt leukemics and in normal subjects, but was significantly lower than in normal subjects.

Price et al[13] have shown that normal adult neutrophils release three different types of CSA molecules and that neutrophils from overt leukemics release only one of the three molecules with CSA. Senn et al[14] showed this same abnormality in three of six patients with idiopathic sideroblastic anemia. Two of these three patients eventually developed acute leukemia, while the third died of a myocardial infarction after a very short period of followup. The three patients with a normal spectrum of molecules with CSA have not become leukemic.

Golde and Cline[15] have used bone marrow cells grown in liquid culture medium rather than soft agar in their studies of overtly leukemic and preleukemic subjects. They have found that overall cell proliferation as measured by labeling index (the ability of the cells to incorporate radioactive thymidine during cell replication — a measure of the growth fraction of cells) falls

Table III

FINDINGS OF BONE MARROW CHROMOSOMAL ANALYSIS AND CULTURE TECHNIQUES IN PRELEUKEMIA AND ACUTE LEUKEMIA

	Normal Subjects	Preleukemia Patients	Acute Leukemia Patients
Chromosomes			
Abnormal	<10%	~25%	~50%
Survival time of patients		↓ with abnormal chromosomes	↓ with abnormal chromosomes
Bone Marrow Culture			
Colony Forming Capacity	Normal	↓↓	↓ or abortive colonies
Colony Stimulating Activity (CSA)	Normal		↓ ↓ ↓
No. of Different CSA Molecules Secreted	3	1	1
Labeling Index	Normal	Low	Highly variable (low-high)*
Maturation of WBC in Liquid Culture	Normal	Little	Essentially None

*A high labeling index in acute leukemia signifies rapid cell replication and in general is an indication for intensive antileukemic therapy.

within the normal range in bone marrows cultured from both preleukemic and overtly leukemic subjects, but that in both leukemics and preleukemics progression beyond blasts to mature leukocytes is significantly slower than normal.

A summary of the findings of these new laboratory tests in investigating leukemia and preleukemia is presented in Table III.

COMMENTS

These new tests appear to hold great promise, both as reliable means of identifying the poor-prognosis patients and as research tools in the study of the preleukemic syndrome. Although these tests are not yet widely available, many centers, including the University of Arizona Medical Center, are now capable of performing these studies in limited numbers of patients and physicians should consider them in the diagnosis of difficult cases.

Currently, there is no specific treatment for preleukemia. Neither androgens nor corticosteroids affect the various cytopenias and there is no evidence to date that "prophylactic" treatment with intensive antileukemic therapy prevents the occurrence of acute leukemia or alters the natural history of the preleukemic syndrome. In fact, intensive cytotoxic therapy may even shorten the survival of preleukemic patients. Although an argument for early intervention is being made by Pierre and others and attempts to initiate an international preleukemia study are underway, it is our feeling that patients with the preleukemic syndrome have such a variable natural history that early treatment with intensive antileukemic therapy is not yet justified. We feel that these patients should be followed and managed with good supportive care (e. g., transfusions) until acute leukemia develops, at which time appropriate intensive antileukemic therapy should be instituted.

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Obstetrics
and Gynecology

ANESTHESIA FOR OBSTETRICS

DANIEL REMEN, M.D.
DONALD J. ZIEHM, M.D., EDITOR

Obstetrical anesthesia has changed markedly during the last 10-15 years. With the development of new drugs and anesthetic techniques, the concept of "just a little anesthesia" for a delivery is no longer valid. Many deliveries are emergencies, some are life threatening. The obstetrical patient must be treated with the same standards of care as the surgical patient. The same equipment, medications and anesthetic drugs that are available in the general operating room must be provided to mother and fetus. Proper obstetrical anesthesia demands correct equipment, skilled manpower, and the right facility.

Obstetrical practices have changed since the time when deliveries took place at home under truly natural conditions. Today's hospital deliveries are far safer, more controlled, and much more comfortable for the mother. Prior orientation and education of the expectant mother is an integral part of the well managed delivery. The mother who is overexcited, distressed, bewildered, fearful or exhausted by labor has a higher risk of both physical and emotional trauma. Knowledge by the expectant parents of the basic physiology involved in labor and delivery is critical to their understanding of the techniques of pain relief. However, explanations must be tailored to the individual patient, for mothers vary in intelligence and motivation. The distorted facts and "old wives tales" acquired from friends and relatives require clarification. With this knowledge the expectant parents can better understand the different modalities of pain control, and the time and circumstances when each can be used.

Before administering an anesthetic to an obstetrical patient, her general medical history and physical condition must be assessed. Blood pressure readings are correlated with the patient's position. A clue to the possible development of the hypotensive syndrome of pregnancy can be gained by obtaining a blood pressure with the patient on her back. *Communication with the obstetrician* should cover the patient's state of health, history of the current pregnancy, the anticipated labor and delivery, and the degree of obstetrical risk involved. His estimation of the infant's size and maturity are very important. With all this information the methods of anesthesia can be planned. Available methods include paracervical block for first stage; parenteral narcotics, lumbar epidural, general analgesia and hypnosis for first and second stage; low spinal block, general anesthesia, caudal and local for second stage only. Most obstetric anesthesiologists consider the continuous segmental lumbar epidural to be the "champagne" of obstetric anesthesia. Conduction anesthesia is utilized in 60-80% of deliveries in most large obstetrical centers, while general anesthesia is rarely used.

The woman in labor is considered by many to be the "third sex." Many anatomic, physiologic and dynamic changes pertinent to the anesthesiologist occur during labor and delivery. Cardiovascular and metabolic functions are changed. The patients' reaction to drugs may be altered. Primary considerations are capillary engorgement in the respiratory tract, progressive rise of the diaphragm compensated by increased anteroposterior and transverse diameters of the thoracic cage, increased renal circulation, decreased $PaCO_2$ and increased PaO_2, lung volume changes with marked decrease in functional residual capacity, decreased total peripheral resistance, increased vein distensibility, increased cardiac output, increasing pulse pressure with no change in blood pressure. The gravid uterus presses on the aorta and vena cava producing impaired venous return to the heart and obstructing blood flow to the uterus and fetus. During a uterine contraction approximately 200 ml is added to the central blood volume. Decreased capacity of subarachnoid and extra-dural spaces due to venous engorgement and the exaggerated lumbar lordosis of pregnancy is present. Additional changes include increased glomerular filtration rate, rise in plasma volume and white blood cells, rise in cerebrospinal fluid pressure during uterine contractions, and an altered reaction to drugs caused by hormonal and physiologic changes during pregnancy.

Lumbar epidural. A continuous lumbar epidural block performed with the correct concentration of local anesthetic (2% Nesacaine or 0.25% Marcaine, adequate to anesthetize the small fibers of the sympathetic nervous system) will result in a block of the afferent pain fibers

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Presented at Maricopa County General Hospital September 7, 1976.
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without appreciably affecting the motor activity of the abdominal muscles. The patient remains awake and can cooperate by pushing when requested to do so in the second stage. Prehydration with 300 ml of electrolyte solution will usually compensate for the vasodilatation caused by the segmental sympathetic block. To avoid hypotension a wedge is placed under the patient's right hip to dislodge the uterus from the vena cava and aorta. Epidural anesthesia has often been observed to improve an uncoordinated labor, an action attributed to the change in catecholamine balance which follows the blocking of their pathways. A large amount of epinephrine may be secreted by the adrenal gland in an apprehensive mother, thereby decreasing the amplitude and frequency of uterine contractions.

Although the benefits of lumbar epidural are many, there are risks involved. Allergic and even toxic reactions can occur. Cardiovascular and respiratory side effects are always possible. The patient is assured that these problems can be managed if they should occur.

Paracervical Block is associated with higher blood levels of local anesthetic in mother and baby. Fetal bradycardia and cardiac deceleration, though usually transient, have been observed with many different anesthetic agents. Fetal heart rate changes may occur more readily in the acidotic fetus. A high concentration of Lidocaine may constrict the uterine artery and decrease blood flow.

General Anesthesia and Analgesia. Full precautions including intravenous infusion and blood pressure monitoring must be taken. Properly trained personnel and adequate equipment must be available to support respiration and to deal with acute cardiovascular problems if they arise. Regurgitation and aspiration of stomach contents is a very serious potential problem. Stomach emptying is delayed in labor, and the cardiac sphincter is also incompetent, thus making regurgitation a definite threat. From 60-400 ml of fluid may be in the stomach in the laboring mother. If the patient regurgitates and aspirates more than 25 ml of fluid with a pH of 2.5 or less, a chemical pneumonitis will develop, lung tissue will be "burned out," bronchospasm will occur, and hypoxia develops. Administration of 30 ml of antacid every 2-3 hours will decrease the pH of the stomach contents to 3-4 or more. Because a rebound may occur, it must be repeated every 2-3 hours.

Caudal Anesthesia should be used only in the second stage. More anesthetic agent is required than for lumbar epidural to achieve the same T10 level of pain control, significant perineal and abdominal muscle relaxation are produced, and these factors may alter descent of the presenting part. Some degree of paralysis of the lower extremities also occurs.

Spinal Block. Hypotension and post-spinal headache are the most common complications. Hypotension can occur if the gravid uterus presses on the vena cava or if the sympathetic block reaches a high thoracic level. "High spinal" with decreased breathing is probably caused by hypotension, a possibility with even the usual 6-8 mg dose. If hypotension develops, the medullary centers may become hypoxic, and the drive to breathe may be decreased. If the patient stops breathing, raise the legs, displace the uterus to the left, and give Ephedrine 10-15 mg IV. The blood pressure will increase, the medulla will be adequately perfused, the hypoxic insult will disappear, and the patient will begin to breathe again spontaneously. It is virtually impossible to paralyze the diaphragm with 6-8 mg of Pontocaine. C4 is a large nerve and difficult to paralyze. 80-90% of respiratory effort is accounted for by excursion of the diaphragm.

Hypnosis. Pain is a complex experience of which fear and apprehension are major components. Hypnosis is a valuable tool with which to reduce apprehension and fear by decreasing the patients perception of the pain of labor and delivery. Patients readily recognize the benefits of adequate relaxation. Various methods of induction may be used. Developing hallucinations and reinforcing them are hard work for all concerned. Specific suggestions for pain control can also be used. The power of positive suggestion is surprising. Not only is relaxation improved, but the patients' attitude to her labor improves and the course of labor and delivery may also be more pleasant. Mothers guided through labor and delivery in this manner tend to produce babies with higher Apgar scores, and the total drugs given are reduced.

The goal of the obstetric anesthesiologist is to allow the mother as comfortable and safe a delivery as possible, with the safety of mother and fetus as the prime responsibility. The administration of the appropriate anesthetic technique when needed, and minimizing the use of medication which crosses the placenta and depress the fetus are important to obtaining that goal.

Seminars in Endocrinology and Metabolism

OSTEOPOROSIS: AN UNRESOLVED PROBLEM

MARSHALL B BLOCK, M.D., EDITOR

Continuing with the present issue of Arizona Medicine is the series of articles entitled "Seminars in Endocrinology and Metabolism." The purpose of these short review articles is twofold. First, due to the rapid proliferation of new knowledge in the field of endocrinology and the multiple tests available for their evaluation, short, clinically oriented reviews would enable the physician to keep abreast of these newer developments as they relate to their practice. In addition, with great stress being placed on voluntary recertification in many subspecialties, reviews such as they could serve as an authoritative, succinct teaching forum. The editors will endeavor to accomplish these goals by utilizing the talents of practicing physicians as guest contributors to this series. Feedback, both positive and negative, is encouraged in order to help us fulfill these objectives.

Our lack of understanding of the underlying nature of osteoporosis is reflected in the large number of clinical papers searching for the optimal treatment for this condition. A unified concept of bone physiology which can incorporate what we know of bone histology, mineral turnover, hormonal influences and physical stresses is not presently at hand. Without such a basic foundation it is little wonder that our attempts to treat this disorder has led to a great deal of frustration for both the physician and patient. Furthermore, the classification of this disorder is artificial and without a rational basis. This in turn has lead to a great many "empiric" and at times "sensational" approaches to the treatment of this disorder. It is not surprising then that orthopedists, osteopaths, internists, physical therapists, nutritionists and chiropractors have a hand in caring for patients with this condition.

Recently a great deal of effort has gone into quantifying bone in human subjects in order to find a measure to follow in utilizing the various modalities of therapy that are presently available. These measurements have included x-ray measurements of cortical bone width, especially in long bones,[1] radiodensitometry by comparison with graded standards,[2] and most recently, absorptiometry.[3] However, all of these methods are

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not sensitive to the small changes in ionic flux which do take place at bone reabsorptive surfaces. Thus, measurements utilizing these techniques lack the sensitivity necessary to detect small changes in mineral content. The changes which take place in ionic metabolism such as those of calcium and phosphorous are furthermore very difficult to measure. This is because calcium balance includes not only what we take in but also what we excrete. The excretion of calcium from the body takes place not only through urinary calcium loss but also via the gastro-intestinal tract. For instance, the majority of calcium taken in by mouth is not absorbed but is excreted in the stool. Furthermore, endogenous calcium coming from bone is excreted into the GI tract and is, thus, included. It is possible that fecal calcium loss may be an important factor in patients with osteoporosis, however in many reported studies it has not been measured. To complicate matters further, there are changes in the normal excretory pattern of calcium which occurs with advancing age, i.e., increased fecal calcium loss occurs in older patients. Therefore, decreased dietary absorption of calcium seems to be a common finding in elderly patients.

Conflicting findings in the hormonal influences which accompany osteoporosis have been reported. For instance, high or normal levels of serum parathormone in this condition have been noted. Likewise, endogenous levels of serum calcitonin have been normal. However, measurements of these compounds by radioimmunoassay methods is open to some criticism inasmuch as there are multiple forms of these hormones circulating in the blood and it is very difficult to know with certainty which fragments of the active hormone are actually being measured. Most early studies of parathormone and calcitonin levels in this condition are thus difficult to interpret.

A form of osteoporosis which has been under scrutiny for many years is that which occurs in patients in the post-menopausal years. However, there are conflicting reports in the literature concerning longterm trials of estrogen therapy on osteoporosis. Some studies have shown some improvement in the condition while others haven't. It is thus somewhat difficult to draw definitive conclusions although most authorities would recommend that patients in the post-menopausal years be treated with estrogen. However, this recommendation needs to be buffered by recent studies which have brought to light significant side effects of chronic estrogen therapy, i.e., relationship to breast and uterine cancer. Furthermore, there are many women who are post-menopausal who do not develop osteoporosis and thus the role of estrogen deficiency may not be very important. The terms "relative estrogen deficiency" as well as some form of "increased requirement" in the patients who do develop osteoporosis is said to be responsible for the beneficial effect of estrogen that is observed.

Of immense interest but little studied are the effects of physical stress on bone remodeling and their effect on osteoporosis. The weight-bearing functions of the skeleton has considerable effect on the remodeling process which takes place in long bones as well as the bones of the vertebral column. This is a variable which is very difficult to control in studies aimed at finding the etiology and treatment of osteoporosis.

Adding further confusion to the above possible abnormalities in causation of osteoporosis is the fact that this condition can sometimes spontaneously resolve. Thus studies aimed at showing the efficacy of one form of therapy over another can be difficult to interpret in light of these naturally occurring remissions. Nevertheless treatment of an individual patient with this condition includes the use of oral calcium supplements, vitamin D preparations and estrogenic compounds. The results to date in this condition are not straightforward, however, noted authorities continue to recommend combinations of these various agents. Recently the use of sodium fluoride as an additional adjuvant to the above has been advocated by one group in particular, however, the results have not been independently verified.

In summary, osteoporosis is a disabling condition affecting many women in the post-menopausal years. The treatment of this condition is very difficult and at the present time completely empiric, because we do not know the true nature of underlying bone physiology. Although there are advocates of therapy with vitamin D, oral calcium, estrogen and sodium fluoride, there is no consistent agreement among authorities about the efficacy of these various agents. Further advances in this field must await a better understanding of bone physiology.

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Seminars in Chest Medicine

CLINICAL STAGES AND DIAGNOSTIC STANDARDS FOR HUMAN COCCIDIOIDOMYCOSIS

CARL F. DIENER, M. D.

This continuing series of articles entitled "Seminars in Chest medicine" will attempt to keep the reader abreast of developments in the broad field of pulmonary diseases. The format used will be that of brief succinct reviews written by the editors as well as guest contributors. Areas of controversy as well as practical chest medicine will be explored. We hope that these reviews will be of value in promoting continuing education for certification examinations as well as a forum for new and controversial issues. The editors welcome comments and discussion from our readers.

ROBERT J. CLARK, M.D.
LYNN M. TAUSSIG, M.D.
WILLIAM C. WEESE, M.D.

The following statement of the Clinical Stages and Diagnostic Standards for Human Coccidioidomycosis was formally adopted by the Arizona Thoracic Society on May 8, 1976. Because it represents the official concepts of the society, we are pleased to publish it in our seminars. We welcome comments from the readers of Arizona Medicine on this important issue. If sufficient response is obtained, a summary will follow in a subsequent issue.

The Editors

Arizona is unique in the United States because more than three quarters of its population reside in areas endemic for coccidioidomycosis. Realizing this, the Arizona Thoracic Society has begun efforts to improve recognition and management of this illness. The first undertaking in this direction has been the development of a staging and diagnostic standards scheme capable of providing a more substantial basis for communications regarding the disease.

More than two years ago, work was begun to formulate a practical classification for human coccidioidomycosis. Several times during the development, suggestions from the 177 members of the Arizona Thoracic Society have been solicited. Opinions from subspecialists interested in coccidioidomycosis from outside of Arizona have also been solicited, and we are grateful for the responses received. At its recent annual meeting, the Arizona Thoracic Society formally adopted the *Clinical Stages* and *Diagnostic Standards* for *Human Coccidioidomycosis* (see table).

These results are conceived only as a beginning with the hope that future accumulations of clinical data and evolving immunological techniques will add refinements and precision to the understanding and description of this illness. As adopted, this scheme considers the extent, the progression and the persistence of coccidioidomycosis, and the associated morbidity and possible mortality, as well as infection without these aspects.

Generalizations for therapy relative to these various stages of illness had been considered, but therapeutic guidelines have not been included. This is because precise guidelines for all stages are not yet clear, and any guidelines would require modification as available therapy changes.

Although not included in the officially adopted *Stages and Standards*, generalizations can be offered. Antifungal therapy seems unnecessary for primary and uncomplicated residual lesions, but would seem indicated for progressive infections. Our first challenge is the early recognition of progressive infection (either pulmonary or disseminated) apart from a prominent primary infection. Fortunately, prompt clinical and serological regression predominates in the general population which acquires the primary infec-

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tion. The remaining cases pose a challenge; clear distinction requires careful analysis of collected clinical data and hopefully will be aided by anticipated discoveries in immunological methods.

The unique stress of the *Stages and Diagnostic Standards* given to primary infection in patients known to have impaired or suppressed immune capacity has special modern relevance because of the wider application of drugs known to impair immune capacity. Currently the therapeutic considerations for this particular combination of

Table I

Clinical Stages and Diagnostic Standards for Human Coccidioidomycosis

Stage	Standard	Remarks
Coccidioidin Reactor	Definite induration more than 24 hours after injection of coccidioidin antigen.	This demonstrates cell mediated hypersensitivity to this antigen.
Primary Pulmonary Coccidioidomycosis	Recent appearance of either a lung parenchymal infiltrative lesion on chest radiograph or a non-localized erythematous skin eruption. Either must be associated with a serological test positive for coccidioidomycosis, isolation of *C. immitis*, or documented conversion of coccidioidin skin test.	Coryza is uncommon in the associated flu-like syndrome. The precipitin test is positive only during and for the few months after this primary stage.
Progressive Pulmonary Coccidioidomycosis	A lung lesion which is worsening or at least not improving over a 1-3 month observation period associated with either isolation of *C. immitis* or rising serological titres.	Uncommon stage in otherwise healthy people. More common in the immunologically compromised host.
Residual Pulmonary Coccidioidomycosis	Demonstration of a diminished, but persisting radiographic abnormality following the observed primary coccidioidomycosis or the demonstration of *C. immitis* in a resected lung lesion. This is applicable only to the patient meeting neither the criteria for progressive pulmonary nor disseminated coccidioidomycosis.	Small nodular lesions are seen most commonly. Thin-walled cavities are frequent.
Anticipated Disseminated Coccidioidomycosis	Primary coccidioidomycosis in a patient known to have an incompetent or suppressed immune system.	Often applicable to organ transplant recipients or other patients on cytotoxic therapy
Progressive Disseminated Coccidioidomycosis	Isolation or histological demonstration of *C. immitis* from a patient with meningitis or with a progressive nonpulmonary lesion.	Complement fixing antibodies to coccidioidin in cerebrospinal fluid is accepted as evidence for *C. immitis* meningitis.
Static Disseminated Coccidioidomycosis	*C. immitis* demonstrated as in progressive disseminated coccidioidomycosis but the source lesion must be clinically stable and complement fixing antibody titres not increasing.	Although this is an uncommon stage, joint lesions are a common expression.
Not Stageable	Presence of *C. immitis* or positive serological test in a patient not fitting any of above categories.	Please report case to committee.

patient circumstances include: a) simple withdrawal of the immunosuppressive treatment, b) addition of an antifungal agent for the primary infection, c) following both above mentioned steps, and, d) observation alone, hoping that the existing immune capacity is adequate to suppress the primary infection. Such cases present another serious challange which will only be resolved with data accumulation.

Progressive infections whether pulmonary or disseminated usually warrant suppressive therapy and the only approved drug at this writing is Amphotericin B. Hopefully, better tolerated and more effective drugs are in the offing. Exterpative surgery long used as therapy for local infections will probably continue to find application in selected cases as long as Amphotericin B remains the sole approved antifungal agent.

The *Clinical Stages and Diagnostic Standards For Human Coccidioidomycosis* have been proposed so that within any defined stage the illness encompasses essentially the same range of clinical phenomena. Hopefully, the outline will facilitate the collection of sufficient objective clinical observations to answer the above challenges and to define better the morbidity and mortality associated with the progressive forms of this disease. A further test of the *Stages and Diagnostic Standards* is the inclusion of the "unstageable" or unclassifiable category. This should test the classification design as well as provide an indication of the need for change.

The Department of Health Services of the State of Arizona is now offered a practical basis for soliciting the reporting of selected stages of coccidioidomycosis cases. The Arizona Thoracic Society must continue its dedication to maintain its responsiveness to these challenges and to aid our colleagues in distant places who see coccidioidomycosis only in those who have travelled to our region.

CARL F. DIENER, CHAIRMAN
Coccidioidomycosis Classification Committee
Arizona Thoracic Society, Medical Section
Arizona Lung Association

Renal Disease

ACUTE RENAL FAILURE - I

GEORGE M. NICKAS, M.D.

General Considerations

Acute renal failure (ARF) has been the subject of periodic reviews. The commonly accepted definition is acute, progressive renal failure with a urinary flow rate of 20 ml. per hour or less; however non-oliguric ARF is increasingly recognized with urine volumes of 1 liter or more per 24 hours with an identical clinical syndrome.

The division of oliguric ARF into pre-renal (functional), renal (parenchymal) and post-renal (obstructive) categories still serves a useful purpose. Pre-renal (functional ARF) such as is seen with hypovolemic hypotension and severe congestive heart failure obviously deserves recognition. Without proper treatment what was originally functional ARF may progress to an established form of renal (parenchymal) ARF. Post-renal ARF equally deserves recognition. The history and physical findings, the occasional pattern of fluctuations in urine volume in the absence of diuretic therapy generally alert one to this diagnosis. Appropriate urologic evaluation is important in making the proper diagnosis and relief of the obstruction.

Renal or parenchymal ARF is due to a diversity of conditions. The most common in approximate decreasing frequency are acute tubular necrosis (vasomotor nephropathy), acute post-streptoccal glomerulonephritis, various forms of renal vasculitis, malignant hypertension, renal cortical necrosis. Other less common causes would include hepato-renal syndrome, athero-embolic renal failure and renal vascular occlusion due to renal arterial or venous embolus or thrombosis. Despite the variety of the above conditions, clinical and readily available

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From: 925 E. McDowell Rd., Phoenix, AZ 85006 (Dr. Nickas diagnostic studies generally point to the proper diagnosis without great difficulty. Post-streptococcal glomerulonephritis typically has findings of urinary red cell casts, hypertension and serologic evidence of a recent streptococcal infection with C-3 hypocomplementemia. The renal vasculitis syndromes including hypersensitivity angitis, polyarteritis nodosa, SLE, and others, will usually show marked urinary abnormalities with proteinuria, micro or gross hematuria and occasionally red cell casts. The presence of an acute nephropathy may be the predominant presenting problem, for example, scleroderma. Renal cortical necrosis is a relatively unusual cause of ARF and is usually associated with obstetric catastrophes, hemolytic-uremic syndrome, or sepsis and may not be initially differentiated from acute tubular necrosis. Unlike ATN, renal functional recovery is extremely rare and renal biopsy may be necessary to establish the diagnosis. Renal parenchymal calcification may be evidence radiologically five to six weeks after onset and may be a helpful clue to the diagnosis.

In the oliguria of hepato-renal syndrome the urine sodium concentration is typically less than 20 - 30 mEq per liter and proteinuria is minimal as are urine sediment changes. The associated severe hepatic disease usually points clearly to the correct diagnosis. Athero-embolic renal failure may be suspected in the older age group in the presence of severe abdominal aortic atherosclerosis and calcification with aneurysm formation. Renal failure usually follows catheter manipulation of the atheromatous aorta or postoperatively with aneurysmectomy but may be spontaneous. Renal biopsy findings are diagnostic and should demonstrate the crystal cholesterol material.

Mention should be made of acute ARF caused by hypercalcemia or hyperuricemia. Conditions associated with severe hypercalcemia, i. e., greater than 15 mg.%, and hypercalciuria may lead to rapidly progressive renal failure. Primary hyperparathyroidism, malignancy with skeletal involvement and multiple myeloma are important underlying causes. Therapy with regard to the renal failure requires prompt reduction of the serum calcium level with rehydration and administration of saline solution, intravenous furosemide to promote calcium diuresis, corticosteroids, Mithramycin. Occasionally dialysis is necessary to relieve life threatening hypercalcemia in addition to the above measures. Hyperuricemic renal failure usually occurs with serum acid levels of 20 mg% or more and is the subject of a recent excellent review. Serum urate levels of greater than 20 mg% are distinctly unusual in acute renal failure unless complicated by hematologic malignancy. The acute leukemias, both myelocytic and lymphocytic, and malignany lymphomas, either with or without preceding chemo-therapy, account for virtually all reported cases of hyperuricemic acute renal failure. Therapy of this disorder is directed toward prompt reduction of the serum urate levels: rehydration, allopurinol, vigorous diuretic therapy and intensive hemodialysis are important tools of treatment. Urologic study and relief of urate stone obstruction may rarely be needed.

Acute Tubular Necrosis
(vasomotor nephropathy)

The most common cause of parenchymal ARF remains acute tubular necrosis (ATN) or in current terminology, vasomotor nephropathy (VN). The current term VN reflects renewed interest in the altered renal hemodynamics rather than the relatively minor histologic tubular changes of degeneration which in fact may be absent. Measured total renal blood flow in VN decreases to approximately one-third of normal and importantly renal cortical arterial blood flow decreases to negligible levels. These ischemic changes may be due to increased intra-renal activity of the renin-angiotensin system. However short term intra-renal infusion of furosemide, which possesses vasodilator as well as diuretic properties, in patients with oliguric ARF due to VN has failed to demonstrate improvement in either total renal or preferential renal cortical blood flow. No improvement in renal functional recovery can be demonstrated.

The causes of VN are numerous. The common causes are nephrotoxins including a variety of therapeutic and diagnostic agents, shock, sepsis, intra-vascular hemolysis and trauma. In many instances a cause cannot be clearly indentified. Despite the complexities of pathogenesis and management of VN, several generalizations are in order. Oliguria with urine sodium concentrations of 40 mEq per liter or more and urine osmolality of 350 milliosmos per liter or less are characteristic urinary findings. High output or non-oliguric VN occurs more rarely than the oliguric variety but urinary isotonicity and progressive renal failure and its sequelae occur as with the oliguric form. Importantly VN is a reversible form of renal failure although oliguria may persist for up to six to eight weeks. The general period of oliguria is approximately two weeks. Renal functional recovery is usually rapid with advent of the diuretic phase. The overall mortality of VN despite improvements in understanding and management remains high at 50% and for certain categories such as post-operative and post-traumatic VN, mortality rates approach 65 - 70%. Finally unless the period of oliguria is extremely brief, that is less than four to five days, and complications can be effectively and readily controlled, dialysis, either peritoneal or extracorporeal, is an essential tool in treatment. Unfortunately there are no reliable clinical or laboratory indicators which can predict the length of oliguric phase so that virtually all patients with oliguric VN must be initially considered as possible candidates for acute dialysis.

Mention should be made of the use of diruetics in the use of acute renal failure. Diuretics such as mannitol, ethacrynic acid and furosemide probably have their most useful role in differentiating functional ARF from VN. In the patient with functional ARF who is not severely hypotensive or hypovolemic the use of these diuretics may prompt an increase in urinary flow rate. Mannitol in the form of bolus doses of 12.5 to 25 grams or infusion of 10% solution, or furosemide in increasing doses up to 500 mg. intravenously may be used for test purposes. If a diuretic response of 50 ml. per hour or more is obtained, then intermittent or drip infusion diuretic may be used to sustain the diuresis. In general however patients with established VN will not respond to these vigorous diuretic measures. Only rarely will a patient with oliguric VN convert to a "high output" state. If a diuretic response is not forthcoming with two or three doses of the above diuretics, they should be discontinued, since complications of hypervolemia from mannitol or deafness from furosemide or ethacrynic acid are potential hazards. Once oliguric VN is firmly established, these agents have no role. The study of Muth with furosemide in ARF failed to demonstrate any therapeutic benefit except in that category of patients with functional renal failure due to cardiogenic shock and cardiac decompensation.

The management of vasomotor nephropathy will be discussed in a subsequent section.

Grand Rounds

PROGNOSIS OF MYOCARDIAL INFARCTION FOLLOWING HOSPITAL DISCHARGE

JAY W. SMITH, M.D.

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CASE REPORT: The patient today was presented by Dr. Lewis Wesselius (medicine resident).

Mr. W. is a 72 year old man with mild emphysema, but no known prior cardiac disease. Two weeks prior to admission, he began having episodes of chest pain. On the day of his admission the patient developed severe chest pain with radiation to both arms, associated with nausea and some dyspnea. Physical examination was normal except for a three component pericardial friction rub. The electrocardiogram (EKG) showed right bundle branch block (RBBB) and changes consistent with acute anterior myocardial infarction. The cardiac enzymes were also consistent with an evolving myocardial infarction.

A temporary pacemaker was inserted because of RBBB in the setting of an acute anterior myocardial infaction. The patient was noted to have an increased number of premature ventricular contractions (PVC's) which were poorly controlled with an intravenous lidocaine infusion. Procaine amide was subsequently added without good suppression. The pacemaker wire was removed and reintroduced with little change in the frequency of the PVC's.

Five days after admission the EKG showed P-R

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prolongation, left axis deviation and RBBB. A ventricular gallop was noted and digoxin was started. Eight days after admission a permanent pacemaker was placed. Subsequently there was again increased difficulty with PVC's. Attempts were made to control the PVC's with several antiarrhythmics including lidocaine, procaine amide, and Dilantin. An attempt was made to overdrive the ectopic foci by increasing the rate of the pacemaker to 100. Propranolol was tried in addition with resulting evidence of clinical deterioration felt to be secondary to diminished cardiac output.

Ten days after admission the patient developed ventricular fibrillation, and he was defibrillated to a paced rhythm. Urinary output was noted to diminish following the arrest. A Swan-Ganz catheter was inserted and the pulmonary wedge pressure determined at 35 mmHg with cardiac output 2.8 liters per minute. The patient was started on a nitroprusside infusion which reduced the wedge pressure to 25 mmHg but with no change in the cardiac output.

Over the next several days the patient had continued PVC's and an episode of ventricular tachycardia requiring cardioversion. He also developed further clinical and radiographic evidence of congestive heart failure.

Nitroprusside was discontinued and diuretic therapy was initiated. The patient had a good response with clinical improvement and further decrease in the pulmonary artery diastolic pressures. Over the following days arrhythmias diminished and the patient was started on quinidine.

Twenty-one days after admission the patient was transferred to the post coronary care unit and was slowly, progressively ambulated. Medications at that time included digoxin, furosemide, quinidine and sublingual Isordil. PVC's were noted to be considerably diminished.

A exercise test to 60% predicted maximal heart rate showed only rare PVC's and nonspecific ST-T wave changes. The patient was discharged two days later, 33 days after admission.

Dr. Smith:

Certainly a reliable estimate of prognosis after an acute myocardial infarction would allow the patient and family to determine future plans and would allow the physician to plan future management. Equally important is identifying patients of equal risk so that therapeutic interventions can be properly evaluated. The therapeutic intervention must be applied to an experimental group with a prognosis equal to the control group. The intervention will have its greatest beneficial effect in a group of high risk patients so efforts should be made to identify such patients. During these rounds those factors which confer high risk on patients and those therapeutic measures which have been used to decrease this risk will be discussed.

The course after myocardial infarction has been divided into the pre-hospital, hospital and post hospital phases. The pre-hospital phase is defined as the time between onset of symptoms and arrival at the hospital. The mortality during the prehospital phase is 25%, during the hospital 15%, and 10% during the first year after discharge from the hospital.[1] I wish to look at the third phase, the post hospital phase. The problem is simply: Can patients with increased risk be identified? More importantly, can the course of these patients be modified?

Before cardiac monitoring was universally available, R. M. Norris, et. al.[2] constructed a coronary prognostic index assessing the relative importance of each factor by the method of discriminant analysis. Of numerous factors investigated 4 were relevant to 3-year survival: pulmonary edema, age, previous myocardial infarction and cardiomegaly. Sex, obesity, diabetes, hypertension, position of the infarct and systolic blood pressure on admission to the hospital were assessed but not found to be predictive of 3 year survival. The 3 year mortality with an index less than 3 was 12% and greater than 85% if the index was greater than 12. Certainly, Mr. W. the patient presented today, has a poor 3 year prognosis for his coronary prognostic index was 12.3 protending a 85% mortality rate.

One of the important spin-offs from the Coronary Drug Project, a multicentered study from the National Heart and Lung Institute to assess the effectiveness and safety of lipid lowering drugs in prolonging the life of survivors of documented myocardial infarction, was the association of VPC's with mortality.[3] During a average 3 year follow-up the mortality in men with VPC's was 21.4% compared to 11.2% in men with no VPC's determined on a standard 12 lead EKG done during the first office visit following hospital discharge for myocardial infarction. The mortality was 21% in patients with 1-9 VPC's/min, 25% in patients with greater than 9 VPC's/min, and 40% in men with pairs or runs of VPC's. There was a higher mortality in those with a short interval between the peak of the sinus beat T-wave and the R-wave peak of the VPC, but this only approached statistical significance. The EKG with multiformed VPC's carried no increased risk; neither did the origin of the VPC.

Arthur J. Moss, et. al.[4] also were able to identify patients with increased mortality risk by documenting VPC's on a six hour continuous EKG tape recorded within three days of contemplated discharge. Multiple variables were entered into a step wise descriminant analysis program to obtain the most accurate separation of those who survived and those who died from heart related causes during a average follow up of two years. The results from the analysis performed on 16 variables from the entire population indicated significant predictive valve for certain characteristics of VPC's: Pairs and runs of VPC's, the prematurity of the VPC, and the frequency of VPC's. The age of the patient was also of predictive value. These high risk VPC's coupled with the patients age allowed Moss to predict survival or mortality with 91% accuracy.

Similar data have been reported from the University of California (Davis) in patients monitored continuously for 10 hours about 11 days after discharge from the coronary care unit.[5] All patients who died suddenly within a 25 month follow-up had VPC's recorded on the ten hour monitor prior to hospital discharge. There were no sudden deaths in patients without ventricular arrhythmias. Interestingly, ventricular arrhythmias which occurred in the coronary care unit were not predictors of sudden death after the patient was discharged from the hospital for they occurred with equal frequency in survivors and non-survivors. Likewise, coronary risk factors, factors which certainly define a greater risk for developing myocardial infarction, did not descriminate between survival and sudden death in the post hospital period.

Does the exercise treadmill stress test provide anymore information when done just before hospital discharge? Ericsson and his colleagues in Sweden studied 100 patients with myocardial infarction by doing stress tests on them immediately before discharge.[6] During a three month follow-up they found a 10% (2 of 19) mortality in patients who had exercise induced VPC's and 2.5% (2 of 81) mortality in patients with no exercise induced VPC's. The patient under discussion today did have rare VPC's induced when exercised to 60% maximal heart rate.

Shaeffer, et. al.[7] reported ischemic ST depresseion in 7 of 32 active military personnel exercised at 9-10 months post infarction and recorded 5 cardiac complications including one sudden death in a ten month follow-up. The group who did not develop ischemic changes with exercise did not have any subsequent mortality, recurrent infarction or progressive angina. Although a 60% maximal exercise stress test has been useful in the management of patients at Arizona Medical Center, it has not been predictive of post hospital mortality during a 21 month follow-up.[8] The mortality rate in patients with ST and T wave changes induced by exercise was 11.1% (4 of 36) and the mortality rate in patients who did not have any further ST and T wave changes with exercise compared to a resting EKG was 7.7% (2 of 26). Although in this study 23% of the patients developed exercise induced VPC's, none who subsequently died developed VPC's with 60% maximal exercise. Mr. W. did not develop further ST and T wave changes with exercise.

It seems reasonable to assume that continuous monitoring might be a more sensitive method of detecting VPC's than exercise treadmill testing. This was the case as reported by Fitzgerald and DeBusk in 15 patients 3 weeks after a myocardial infarction who were exercised tested and also monitored while ambulatory for 10 continuous hours. [9] Ventricular ectopic activity occurred in 4 patients during treadmill testing and in 13 patients during continuous Holter monitoring. Ryan, et. al.[10] compared the incidence of VPC's during continuous monitoring and exercise testing in 100 unselected patients with coronary artery disease and found continuous monitoring the more sensitive test.

It is also reasonable to assume that prognosis should be worse in patients who have suffered more than one myocardial infarction since cardiac muscle mass must decrease after each infarction. Some studies support an increased mortality after a second infarction[2,11] while others do not.[5] In the Arizona Medical Center study a history of previous myocardial infarction and pulmonary congestion were significantly more common in patients who died in the post infarction period.[8] Schulze, et. al.[12] looked at both ventricular arrhythmias and left ventricular ejection fractions in 81 patients two weeks following acute infarction. Although they projected a one year mortality of 35% in patients with an ejection fraction less than 40%, all 8 patients in the seven month follow-up period who had ventricular fibrillation or sudden death had both an ejection fraction less than 40% and complicated ventricular arrhythmias; none were in the subgroup with an ejection fraction less than 40% and an occasional VPC. They concluded that while a low left ventricular ejection fraction may suggest a poor long term prognosis following myocardial infarction the presence or absence of frequent, multiformed, pairs or runs of VPC's determines the risk of sudden cardiac death in the early post hospitalization period.

Conduction distrubances during the course of acute myocardial infarction have also been found to influence post hospital survival. R. A. Waugh, et. al.[13] found a 3% incidence of sudden death in one year in patients who developed either right bundle branch block (RBBB), left anterior hemiblock (LAHB), or RBBB and LAHB. In patients who developed periinfarction block, left posterior hemiblock (LPHB) left bundle branch block (LBBB), or incomplete left bundle branch block during their infarction the one year incidence of sudden death was 14%. The mortality rate was 45% if the patients developed RBBB and LAHB and P-R prolongation, LBBB and P-R prolongation, RBBB and LPHB, or had Mobitz II progression. It was these data that convinced the physicians caring for Mr. W. to insert a permanent pacemaker for he had RBBB and developed LAHB and P-R prolongation during the acute phase of the infarction.

What can be done to prevent sudden death after discharge from the hospital following myocardial infarction? Of the factors which seem to be important determinants, heart block and ventricular ectopy are the ones that perhaps can be altered. Although the numbers of patients are small Waugh, et. al.[13] noted a marked increase in one year survival in those patients with Mobitz II block who were treated with a permanent transvenous pacemaker. They suggest that prophylaxis against sudden death may be offered by permanent pacemaker therapy and further suggest that this be evaluated in the prospective therapeutic trial.

Procaine amide has been used long-term to prevent ventricular ectopy after acute myocardial infarction.[14] Although the incidence of frequent VPC's was not decreased the incidence of VPC pairs and ventricular tachycardia were decreased significantly. The mortality dropped from 4 of 39 in the control group to 1 of 27 in the treated group—not of statistical significance. Moreover, 23 percent stopped therapy within the first month because of adverse reactions and 54 percent developed adverse reactions. The conclusion of the investigators was that the high incidence of toxicity precludes the long-term prophylactic use of procaine amide.

A recent multicentre international study provides evidence that prognosis following myocardial infarction might be improved with long term beta blockers.[15] Although the drug, practolol, had to be withdrawn because of serious oculocutaneous and peritoneal reactions, the one year mortality following anterior myocardial infarction was reduced significantly. Unfortunately there was no protective effect in patients with inferior infarctions. The lifesaving effects of practolol were almost certainly due to the antiarrhythmic effects of beta blockage and resumably a less toxic drug such as propranolol would have a similar effect. The recommendation from the numerous participants in the study was that all patients receive long term beta blocker therapy after anterior myocardial infarction if no specific contraindications exist. Although there are no similar data with proranolol therapy should be seriously considered.[16]

Other therapeutic interventions such as coronary artery bypass surgery post infarction to prevent sudden death require more extensive trials. Such therapeutic trials are now feasible for high risk patients can be identified.

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MAKING MEDICAL REPORTING LAWS WORK

SUZANNE DANDOY, M.D.
DIRECTOR

Arizona law imposes upon physicians and health care institutions a duty to report their treatment of suspected cases of child abuse,[1] contagious disease,[2] and wounds inflicted by certain weapons.[3] Failure to comply with these reporting laws is a misdemeanor punishable by fines and/or imprisonment. However, despite the stringent penalties that can be visited upon an individual or institution for not reporting the treatment of the conditions described, it is generally acknowledged that reporting requirements of this type are frequently ignored. In a period of increasing awareness of the problem of child abuse and an incidence of venereal disease that has reached near epidemic proportions, the ineffectiveness of reporting laws should be a matter of serious concern.

In attempting to determine what can be done about this problem, several possible approaches have been suggested.

In an article in the American Journal of Public Health,[4] Professor Nathan Hershey argued that physicians and health care institutions that disregard reporting duties should be held civilly liable for any subsequent injuries that might have been prevented if the reporting responsibility had been discharged. The guardian of a battered child, for example, could bring suit against a physician for injuries inflicted by the child's parents after the physician's treatment of the child's injuries, if the physician should have known that the injuries treated were parent-caused.

The courts in two recent California cases have refused to dismiss actions brought against a physician and a hospital for negligence in not reporting an infectious disease and a case of child abuse to the proper authorities.[5] The legal theory on which the courts allowed the cases to go to trial was the same theory as that propounded by Professor Hershey. However, both courts made it clear that while the suits could proceed, the plaintiffs in both instances would have the almost impossible task of proving that the injuries for which damages were sought were causally traceable to the defendant's failure to comply with reporting statutes.

Another approach to the reporting problem is the invocation of disciplinary sanction by the State Board of Medical Examiners. Under Arizona law, the Board has the authority to take disciplinary action against a physician for a list of offenses that are labeled "unprofessional conduct." These offenses include "the commission of a misdemeanor involving moral turpitude" and "conduct . . . which . . . might . . . constitute a danger to the health . . . of the public."[6] Since it is unclear whether either of these definitions are meant to cover a violation of reporting laws, it might strengthen the enforcement of such laws if their violation was explicitly made a form of unprofessional conduct.

The difficulty with both of the approaches discussed above is that they are essentially punitive in nature. They are based upon imposing additional forms of punishment to the criminal penalties already imposed on transgressors of the reporting laws. There would seem to be some question, however, whether adding more,though different types of punishment for breaking an ineffective law can result in making that law any more effective. A strong case can be made for the proposition that reporting laws will only work when physicians recognize that such laws only codify what should be viewed as an ethical obligation.

Physicians usually cite two reasons for not complying with reporting laws.

First, a physician may feel that the reporting of a patient's disease or an instance of child abuse is contrary to the concept of physician-patient confidentiality. The law, however, recognizes the overriding societal benefits to be gained by reporting certain medical conditions and exempts statutory reporting requirements from confidentiality protection.[7]

Second, some physicians fear that a parent with a battered child or a diseased individual may be dissuaded from seeking medical attention if it is known that the condition for which attention is sought may be made known to the authorities. Experience with laws requiring that the treatment of wounds inflicted by certain weapons be reported would seem to indicate that such fears are largely groundless.

In the last few years, the whole area of medical ethics has come in for intense study and debate. Both in medical schools and among the practicing medical profession, issues like the ones discussed here have been the subject of renewed interest. It is in this interest and the acceptance of reporting as an ethical obligation that the real hope of making reporting laws a functioning reality must ultimately lie.

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The A.M.S.A. Report

ON BEING AN M.S. II IN D.C.

This spring, in a specially-designed program—part of a Family Practice "community medicine" clerkship, I travelled to Washington, D.C.

Several factors had prompted this research, most of which concerned experience prior to medical school: experience with many people who received inadequate health care, ranging from Harlem slum dwellers, to college students, to model cities (Tucson) population designing its own community hospital. During the first year basic science frustration, knowing there were rural and urban populations not receiving care, along with seeing little evidence of improving conditions—I became involved in self-made student groups, in addition to the Arizona Medical Association, the American Medical Student Association and eventually the American Medical Association. The same questions were being asked over and over. What are the needs—for which people, what *are* they getting; what *should* they get, who should provide, and why isn't care there?

And over and over, nebulous answers, efforts of organized medicine, theories, some actualities. I decided the most realistic changes would come from the realms of political control in D.C. and that I would see for myself who was making decisions, based on what information, inspired by which ideals and goals. Community medicine agreed, and off I went for three weeks medical school credit in addition to my own week of Spring vacation.

While there I met with senators, representatives, administrators, lobbyists, organized labor, public interest groups, and most importantly, with staff people of Congressman Rogers and Senator Kennedy, A more complete laundry list includes staff member of:

- Senate Committee on Finance Subcommittee Health,
- House Ways and Means Committee Subcommittee on Health,
- Senate Committee on Labor and Public welfare Subcommittee on Health,
- House Committee on Interstate and Foreign Commerce Subcommittee on health and the Environment,
- Senate Committee on Appropriations Subcommittee on Labor, Health, Education, and Welfare.
- Department of Health, Education and Welfare, including Bureau of Health Manpower and National Health Service Corps.
- Office of Technology Assessment,
- AMA, AAMC.
- Institute of Medicine,
- Health Staff Seminars,
- Health Policy Center,
- United Mine Workers,
- AFL CIO,
- Health Research Groups (of Nader fame) and
- Health Policy Advisory Center, New York City.

In addition I attended hearings on influenza vaccine, health manpower, drug package inserts, and the high cost of medical care.

I found that there were some outstanding champions of the the people; congressmen and particularly their staff worked extremely hard (i.e., their hours compared favorably to medicine). There were those who had special interests to protect. The administration had its problems of keeping the budget down while congress was being expansive in its willingness to provide for its constituents.

The pace was frantic, with importance of health legislation ranked very high by some, low by others.

I was impressed that the major emotion pervading much of the decision-making was that doctors had gotten away with too much (money and privilege) for too long, and nobody felt uncomfortable about taking some of the "much" away. Doctors' interests were carefully protected by some powerful members of congress, and seriously challenged by others.

I left with a distinct impression that the practice of medicine will change for various reasons: 1) Spontaneous voluntary actions on the part of the industry; 2) action encouraged by the threat of legislation; and 3) action's legislated.

I was impressed, in addition, with the fact that so much attention was placed on the importance of primary care and how often it was assumed that doctors had the responsibility for it all, while little mention was made of the role of the new health practitioner in providing that care.

People are off in Washington changing the world of medicine by shifting funding, determining critera for Medicare and Medicaid payments, and providing capitation funding for medical schools in exchange for promises of public service.

Sincerity is everywhere apparent; there are many dreams in powerful offices. Unfortunately health care problems are very real and need perhaps more realistic solutions than Washington is willing or able to provide.

Kathryn C. Reed
Class of 1977

Radiology
Case of the Month

CASE NO. 15

PHILIP STRATEMEIER, M.D.
IRWIN M. FREUNDLICH, M.D.

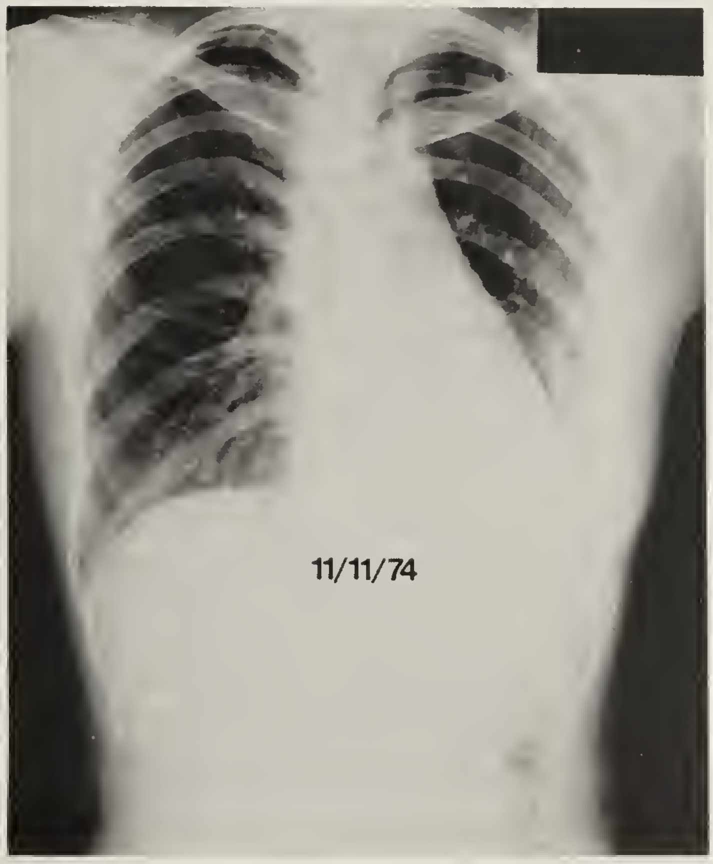

Figure 1

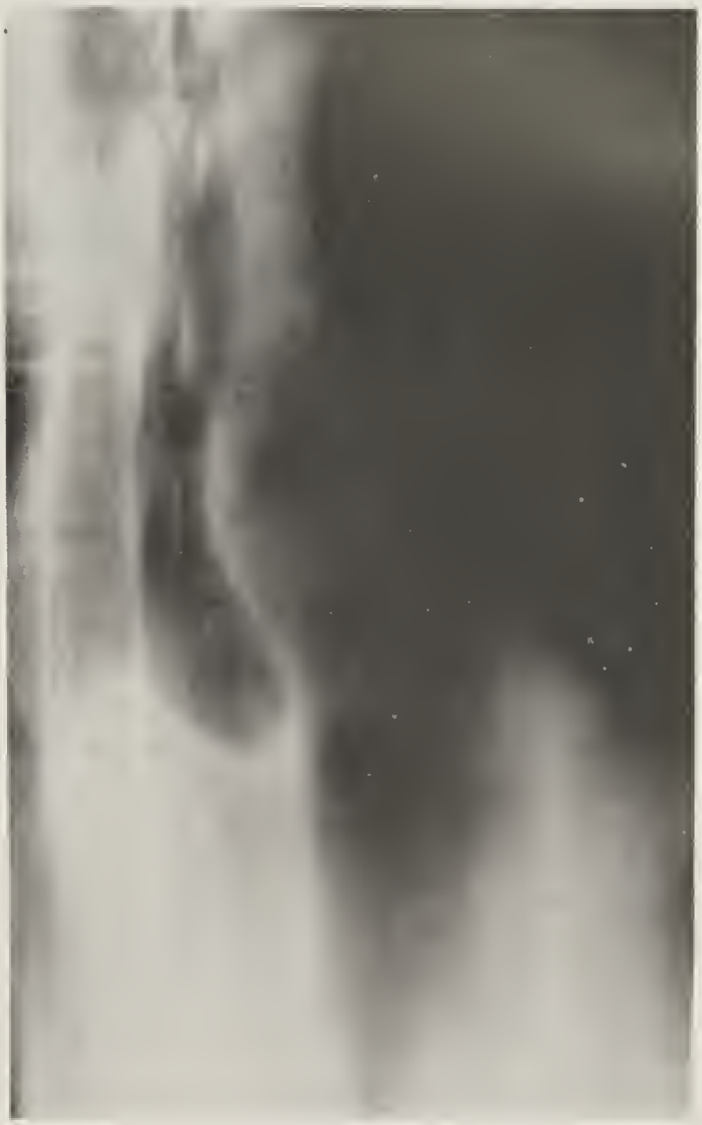

Figure 2

This 26 year old Indian woman presented with increasing shortness of breath, dyspnea on exertion and fatique. Pertinent past history includes cardiac murmurs of aortic disease as well as a motor vehicle accident six months before. Because of the initial radiograph (Figure 1), a tomogram was made of the left hilum (Figure 2). What is your diagnosis?

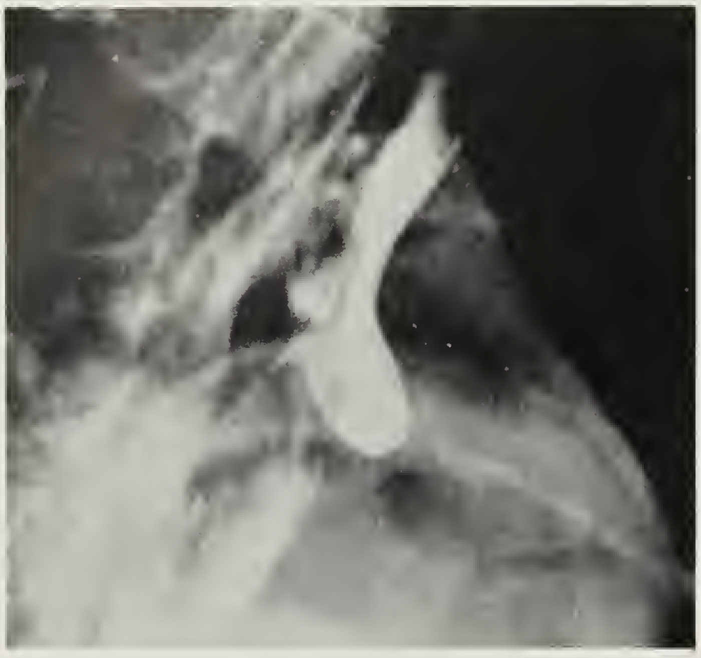

Figure 3

This woman had rheumatic fever at age 3 with variable but not severe symptomatology most of her life. She was hospitalized at another institution after suffering blunt chest trauma in a motor vehicle accident. Although a radiograph at that time suggested collapsed left lower lobe, she signed out of the hospital against medical advice. There was no further investigation until she was seen at this institution with the current symptomatology.

Figure 1 shows oppacification of the left lower lung field silhouetting the left border of the cardiac shadow and left hemidiaphragm, which was secondary to left lower lobe collapse. The tomogram of the left hilum suggest rupture of the left mainstem bronchus as air can be seen in the trachea and left mainstem bronchus where there is an abrupt termination. Figure 3 represents an attempt at bronchography, which confirmed the termination of the bronchus and rotations in multiple positions failed to demonstrate any contrast distal to the termination of the mainstem bronchus. At surgery the left mainstem bronchus was found to be transected with considerable fibrosis presumed secondary to previous hemorrhage. The left lower lobe was totally collpased and eventually the left upper lobe bronchus was anastomosed to the left lower lobe with subsequent re-expansion and ventilation of both lobes. Fractured bronchus is a frequent complication to blunt chest trauma, with the highest incidence in males under 40 years of age.[1] Rupture occurs secondary to acute increased intra pleural pressure from the crush injury, combined with a sudden Valsalva leading to increased intra tracheal pressure.[2,3] The right mainstem bronchus is more frequently affected than the left. Adding to the trauma is the effect of deceleration of the lungs which are fixed at the hilum,[4] leading to stress on the bronchus, though generally there is no effect on the vessels.[5] However, when there is lobar collapse as in this case, the noncollapsed lung is hyperexpanded, leading to spreading of the vascular markings. This causes a relative hyperlucency to the involved side. Indeed, a pulmonary angiogram performed on this patient showed hypoperfusion to the left lung, due to minimal perfusion of the collapsed lobe, and increased resistance in the upper lobe from vascular changes secondary to anoxia.

Bronchial rupture may also lead to pneumothorax and pneumomediastinum often under tension and subcutaneous emphysema. If there is injury to the vessels, then hemothorax can occur. In the present case, there was an associated thoracic vetebral dislocation with associate hemorrhage which led to the fibrosis and pleural changes. Pain, cough, hemoptoyptsis, cyanosis and shock may also result. Frequently the diagnosis isn't made until a month or more later.[6] Repair can be 100% successful as late as 15 years post traums.[7] The current patient had mild transient congestive heart failure post operatively, but otherwise made an uncomplicated recovery.

DIAGNOSIS: Fractured left mainstem bronchus with left lower lobe collapse.

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Topics Of Current Medical Interest

INFORMED CONSENT — RADIOACTIVE WASTE PRODUCTS AND THE ARIZONA NUCLEAR SAFEGUARDS INITIATIVE (PROPOSITION 200)

ANDRÉ BRUWER, M.D., FACR

SUMMARY

The problems of safely isolating radioactive waste products of the nuclear fuel cycle from the environment, have not been solved and remain a potential and actual health hazard of major proportions.

The Arizona Nuclear Safeguards Initiative (Proposition 200) will provide an opportunity for the citizens of Arizona to participate in the educational debate that will precede their November vote.

Physicians must be expected to take part in this educational process.

Voter participation on the basis of such an educational process embodies the principle of informed consent at the social level. This is as it should be in a democracy.

On February 13, 1975, Dr. Robert C. Seamans, Jr., Administrator of the Energy Research and Development Administration (ERDA) said in a speech to the Engineers' Society of Western Pennsylvania, that in order to develop nuclear power we will, among other requirements, have to "demonstrate the capability to safely manage an increasing amount of high level radioactive waste".

This would certainly seem to be a reasonable, rational statement. It is a statement that has been frequently made by apparently cautious proponents of nuclear power plants.

Why is it so important that we be certain that the so-called high-level radioactive wastes be 'safely managed'? The reason is high-level radioactive wastes produced by nuclear power plants, are immensely radioactive and because radioactive substances are harmful to living things, including human beings. All national and international organizations dealing with matters of radioactivity function on the principle that there is no safe level of radiation. This is also called the 'no-threshold principle'.

Experiments performed on laboratory animals to evaluate the possible dangers of inhaled plutonium have resulted in the development of lung cancers in essentially 100 percent of animals tested. Gofman has pointed out in a recent issue of the Journal of the American Medical Association that, in beagles, the infinitesimally small dose of 0.049 micrograms of plutonium 239 per gram of bloodless lung, resulted in lung cancers in 100 percent of the dogs.[1]

About 900 tons of plutonium will have to be reprocessed annually if we are to build 1,000 breeder power reactors. Assuming that inhaled plutonium is as dangerous for humans as it is for beagles, how perfectly would the breeder plutonium have to be contained (this means sealed off from the environment) in order not to be hazardous? Gofman has estimated that even if the industrial containment of plutonium by nuclear power companies is 99.99 per cent perfect, the remaining (escaped) .001 per cent could, as a result of its dispersal in the atmosphere, result in 139,000 lung cancers per year.

There is a tendency to scoff at such estimates. One is sometimes made to feel that the lungs of tough-minded, patriotic and 'responsible' scientists, nuclear engineers, industrialists and others are immune to such nonsense as cancer from a few invisible specks of inhaled plutonium, and that only sissies, nervous Nellies and unpatriotic busy-bodies would get lung cancer from plutonium.

But, in fact, we are always reminded by those officials who have to make responsible decisions and who have to grant licenses to nuclear power facilities, that the high-level radioactive waste products must be isolated from the biosphere ('safely managed' is a common euphemism).

Is it really so difficult to do?

Apparently not, according to those who are in the business of building nuclear power plants.

According to a small brochure distributed to citizens in their energy bills in the Phoenix area by companies participating in the Arizona Nuclear Power Project, "current technology is completely capable of handling the small amount of waste produced by nuclear energy plants".[2]

One almost blushes with shame when one questions such a bland reassurance. After all, responsible people would not reassure the public about the safety of radioactive waste management unless they knew it was true. They would not tell citizens that the educational debate that would be stimulated by an initiative on a popular ballot, is unnecessary, unless they were being totally truthful about their statements on the safety of handling such radioactive waste products.

But things are apparently not as simple as the lobbyists for nuclear power tell us.

Thus, as recently as September 1975, Commissioner Edward A. Mason of the U.S. Nuclear Regulatory Commission (NRC), said "intermediate and long-term management of high-level wastes remains an unanswered technical and policy issue. Simply stated, although several available technologies have been researched, demonstration-scale information is lacking and no agreed-upon plan for long-term, high level waste management exists. And, no matter what plan is eventually proposed, it is quite likely to be the subject of intense public debate not only in the U.S. but also internationally".[3]

This sounds as if the problems of caring for radioactive waste products had not been solved as of September 1975.

As recently as April 15, 1976, Dade W. Moeller, the chairman of the Advisory Committee on Reactor Safeguards of the NRC, reported on recent deliberations concerning the management of radioactive waste products. He stated that "the results of these reviews indicate that the technological development of nuclear waste management has progressed more slowly than desirable considering the growth of the nuclear power industry and the potential public health significance of its associated wastes. Although ERDA is presently planning to implement a broad scale R & D program to correct this situation, the results of this effort are a number of years away. In the meantime, the commercial nuclear power industry and related fuel cycle activities are creating a variety of radioactive wastes whose management and storage are essentially uncoordinated for the purposes of regulatory control".[4]

There would seem to be a vast gap between the facts being provided by officials of the NRC and the assurances which are being given by the officials of the Arizona Nuclear Power Project.

In a very real sense the whole matter of reassurances in regard to safeguarding the health of the people from exposure to radioactive wastes is a form of legalized insanity. Plutonium, for example will remain radioactive for hundreds of thousands of years. Plutonium created by a nuclear power plant today will be as capable of causing lung cancer in infinitesimally small quantities ten thousand years from now as it is right now.

Hannes Alfven, Nobel Laureate in Physics and Professor of Physics at the University of California in San Diego, exposed the madness when he said that "[The nuclear technologists] claim that they have devoted more effort to safety problems than any other technologists have. This is true. From the beginning they have paid much attention to safety and they have been remarkably clever in devising safety precautions. This is perhaps pathetic, but it is not relevant. If a problem is too difficult to solve, one cannot claim that it is solved by pointing to all the efforts made to solve it".[5]

As if to underscore the impossibility of their task, the NRC is now taking the position that we should not delay the nuclear power plant time table because we do not have all the answers to the waste problems. As stated by the Chairman of the NRC, Marcus A. Rowden, on July 12, 1976: ". . . every technical issue [does not] need to be resolved in minute detail in advance of action".[6] The plea is that we should get going and solve the minutely detailed technical problems as we proceed.

Rowden informs us that those who are studying the problems of radioactive wastes have discovered three "time horizons": first, the short-term horizon of five to ten years; second, a horizon of a "few centuries", because history shows that manmade institutions cannot be relied upon to last any longer; and third, a horizon that lies some thousands of years away.

Admitting that we do not yet have the detailed solutions for waste management for the first time horizon, Mr. Rowden stated modestly that, with reference to the third time horizon, "some degree of uncertainty necessarily attaches to predictions that go beyond several thousand years —

uncertainty relating to such factors as demography, climate and earth movements".

Guarding immensely poisonous radioactive materials so that they do not enter our environment in even the minutest quantities, is a deadly-serious business. This the NRC would admit. But to say that we can take risks before we know how to handle all the problems even in the perspective of the first time horizon, sounds irresponsible.

It is like a surgeon telling a patient that his operation will involve three periods. The first, studying about his disease. The second, learning to do the operation, never having done this operation before. And the third, taking care of him in the post-operative period if he made it through the second part. The surgeon then admits that he is not acquainted with the technical details of the operation, but that he is sure he'll master the problems as they arise during surgery.

The difference is that the surgeon has to take out his own malpractice insurance whereas the nuclear power industry has the public to buy its (inadequate as it is) malpractice insurance in the form of the Price-Anderson Act.

So far no scientist or nuclear technologist has solved the riddle of turning off the radioactivity they have turned on. Until they can do this, they should cease playing roulette with the lives and the health and well-being of this and future generations.

Edward Teller, father of the hydrogen bomb and a great proponent of nuclear power plants, has stated that if the voters knew the facts they would be completely capable of understanding the issues that are involved.

The nuclear safeguards initiative will give the people of Arizona an opportunity to discuss and debate the nuclear power plant issue for several months before voting on it in November. It would therefore be a disservice on the part of the power industry to try as hard as possible to eliminate the initiative from the popular ballot. The Arizona Nuclear Power Project and its participating power industries should give the initiative their blessing. They should look forward to the public debate on one of the great and most important issues of our time—and possibly of the times of our great-great-grand children.

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Editorial

THE SICK PHYSICIAN

Physicians are engaged in a demanding profession. Life and death are the responsibilities we have assumed. With every increase in medical knowledge the expectations of the lay public grow apace. The more cures we provide, the more are demanded. And woe and malpractice betide us for even the most human and honest shortcomings.

Since Archaic times we have been a "fraternity". As such, we were admonished even by ancient Hippocrates to give aid, not only to our patients but to our brothers.

And now, very recently, Arizona law reiterates that admonition: ". . . . any doctor of medicine, or the Arizona Medical Association, Inc., or any component county society thereof SHALL, and any other person MAY report to the board—a doctor of medicine (who) is or may be medically incompetent or is or may be guilty of unprofessional conduct or is or may be mentally or physically unable to engage in the practice of medicine. Any person required to report under this section, who provides such information in good faith shall not be subject to suit for civil damages as a result thereof".

The wording is offensively harsh, yet it asks no more than do our ethical principles. We must blame only ourselves, for it is because of our laxity that authority has thus been invoked.

We have tended, even more than laymen do, to close our eyes or overlook or even ignore illnesses or problems in our colleagues and friends. This is not charitable tolerance. It is an abrogation of obligation and responsibility to peers and to patients. It abandons them to their fate—through our rationalizations that we do not wish to do harm. Help was what was needed and we did not provide it. We felt helpless.

The Physicians Rehabilitation Committee's function is to provide that help—before it is too late.

A report to any of its members or to the secretary of Arizona Medical Association will bring prompt assistance and counsel—you will have not only brought timely aid to your colleague but will have met your ethical (and now also legal) responsibility. Timely, in the sense that the committee functions as an intermediary—before the impaired physician has become a problem for the Board of Examiners.

The committee is an arm of your Medical Association. The board is an arm of the law. The committee will of course apprise the board of the problem. The board will take no further action unless the problem has become or threatens to become a legal one.

We therefore urge all physicians, doctors' wives, hospital staffs, administrators, nurses and patients to avail themselves of the committee's services, in behalf of any physician who, whether sick, aged, addicted or otherwise impaired or potentially heading for trouble who has not seen or admitted this problem to himself.

You may write or phone the following committee members:

BENDHEIM, OTTO	5051 N. 34th St., Phoenix	955-6200
BISHOP, WILLIAM	703 Ash St., Globe	425-5759
CLYMER, JOHN	708 N. Country Club Rd., Tucson	793-8365
CRISP, WILLIAM	926 E. McDowell Rd., Phoenix	258-8995
CZERNY, EVERETT W.	1601 N. Tucson Blvd., Tucson	327-5628
DAMSTRA, DONALD	St. Luke's Hospital, Phoenix	258-7373
DUISBERG, RICHARD	1313 N. 2nd Street, Phoenix	254-5161
GREEN, WESLEY, E.	2501 N. 4th St., Flagstaff	774-4374
KAHLE, JOHN F.	715 N. Beaver, Flagstaff	774-7345
LINKNER, LAURENCE	3411 N. 5th Ave., Phoenix	279-7397
SATTENSPIEL, EDWARD	333 W. Thomas Road, Phoenix	264-3267
SCHEIBER, STEPHEN	Arizona Med. Center, U of A	882-6315
TOMLINSON, WALTER	P. O. Box 4, Elgin, AZ	455-5572
VOLDENG, KARL	St. Luke's Hospital, Phoenix	258-7373
WASKOW, ELEANOR	550 W. Thomas Road, Phoenix	266-8236

You may also write or phone Bruce Robinson, Executive Director, ArMA, 810 W. Bethany Home Road, Phoenix, AZ Phone: 246-8901.

Let us be real friends to our friends.

Richard E. H. Duisberg, M.D.
Chairman, Physician Rehabilitation Committee
Arizona Medical Association, Inc.

A POINT OF VIEW

REGIONAL PLASTIC SURGEONS

To the Editor:

At a time when federal agencies and hospitals are especially concerned with definitions, priviliges, and capabilities, it behooves an enlightened medical profession to reflect on who's who in the growing field of plastic surgery. We tend to rely on semantics and impressions. If plastic surgery is needed, we may turn to a plastic surgeon because he is a specialist. That tendency is fine, but in following it, we fail to recognize the many practitioners of plastic surgery who do not use the title "plastic surgeon."

Basically, plastic surgery is both a method and a medical specialty. In effect, the plastic surgeon is a specialist in the method. He is obviously a practitioner of plastic surgery, is certified by the American Board of Plastic Surgery, and is officially designated a plastic surgeon. There is virtually no chance of anyone misunderstanding his scope of medical practice.

Who, then, are the other practitioners of plastic surgery? They are specialists in a particular region or organ of the body, for example, ophthalmologists, who after certification by the medical board in their speciality, subspecialize in plastic and reconstructive surgery. Thus, during their speciality training, they master both the specialty region and the method of ophthalmic plastic surgery. Some of these specialists have become known as regional plastic surgeons.

Unlike general plastic surgeons, regional plastic surgeons do not present a consistent identity pattern. Some otolaryngologists may practice under the title "head and neck plastic surgeon" or "cosmetic facial surgeon." Other regional specialists may add the term "plastic surgeon" to their specialty title, for example, the ophthalmic plastic surgeon and the dermatologic plastic surgeon. Still others, such as orthopedists, obstetrician-gynecologists, urologists, neurosurgeons, colon and rectal surgeons, and general surgeons, do not usually add the term "plastic" to their specialty title, although they may use plastic and reconstructive surgical techniques.

A leading ophthalmic plastic surgeon estimates that 1,000 ophthalmologists do a significant amount of plastic and reconstructive surgery. The chief of one otolaryngology residency training program estimates that 4,000 otolaryngologists perform regional plastic and reconstructive surgery. A prominent dermatologist estimates that 300 members of his specialty practice plastic surgery.

About 180 maxillofacial surgeons spend all their time doing plastic and reconstructive surgery. Virtually all of the neurosurgeons, estimated at 2,000, perform cranioplasties for skull defects. Most of the 9,200 orthopedists practice plastic and reconstructive surgery, because that in the broadest sense is the nature of their work. Many of the 6,000 urologists do some plastic reconstructive surgery in their specialty area, as do many of the 30,000 general surgeons who apply plastic techniques in repairing hernias, strengthening the abdominal wall, removing tumors and ulcers, working with or operating burn units, and repairing defects with skin grafts.

Conservative estimates from the most pertinent medical specialties suggest that about 22,000 American physicians practice regional plastic surgery. Of that total, more than 7,000 perform plastic and reconstructive surgery in the head and neck area alone. The significance of these numbers is seen when they are compared with the number of specialists, an estimated 1,500, who are designated plastic surgeons.

Obviously, no medical specialty has the exclusive capability to perform plastic and reconstructive surgery. No board certification by any specialty and no medical title can guarantee a fine surgical result. In discussing plastic surgery with our patients, we might well remember that there are good, fair, and poor surgeons in all specialties. The most important consideration, therefore, is not the surgeon's title but his ability as manifested by his results.

Trent W. Smith, M.D.
Columbus, Ohio

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In Memoriam

HARRY T. SOUTHWORTH

1904 — 1976

Born in Prescott, June 19, 1904, he was the son of Dr. Harry T. Southworth, Sr. and Fay (Fox) Southworth, who had come to the Territory in 1903. The father was an able, popular family physician and surgeon, and was Prescott City Health Officer at the time of his death in 1933.

Harry T. Southworth, Jr., attended Prescott public schools where he also had a fine record in several school sports. He attended Stanford University, Idaho University and graduated from Rush Medical School, Chicago, in 1933, and obtained an Illinois license in that year. He interned in Washington Blvd.Hospital in Chicago and obtained an Arizona license in 1934.

He married Margaret Orthel in Prescott on August 20, 1931. They had two children, Thomas, of San Jose, California, and Kathryn Ross of Phoenix, and they produced ten grandchildren. His wife preceeded him in death in 1971. He has one sister, Mrs. Tex Ritter, (Dorothy Fay) of Nashville, Tenn. They were members of the Congregational Church. He was a Mason. Not famous as a "joiner", he nevertheless, gave active and effective support to many community activities, especially those related to health and health services.

Long before postgraduate education was popular, or required, Dr. Southworth enrolled in seminars in cardiology, surgery, obstetrics, pediatrics and other fields.

He was on the staff of the Phelps Dodge Hospital in Jerome, Arizona from 1934 to 1938. Coming to Prescott, he was on the medical staff of Mercy Hospital, Yavapai County and Prescott Community Hospitals, and the Yavapai Community Hospital. He served as Chief of Staff several times. He also served as President of the Yavapai County Medical Society, and was elected President of the Arizona State Medical Association in 1951.

During World War II he attended the School of Aviation Medicine at Randolph Field, Texas, and was assigned to the 2nd Bombardment Group of the Army Air Corps, which was stationed at various places in the United States—Nebraska, Washington State and Colorado Springs, Colo.

Poor health and diminishing eyesight, caused him to limit his practice, and finally to retire May 1, 1974, a development that caused widespread regret among his patients, who had found him an able, kindly, dedicated physician, and among his fellow practitioners who enjoyed working with him.

Death occured July 30, 1976, ending a seventy year record of father-son medical practice of the highest professional type. Perhaps a statement made in 1933, by a patient (who also was a member of the state legislature) should be repeated. "Our doctor is dead. Prescott will go on, but it will never be the same without his presence."

ArMA
Medical History

HOSPITALS IN PRESCOTT

FLORENCE B. YOUNT M. D.

August 1976
Prescott, Arizona

Pursuing the history of early hospitals in Arizona, Dr. Yount presents a definitive and most interesting sketch of the Prescott scene. Dr. Florence with her usual reticence has avoided mention of her own extraordinary community efforts in securing the means and methods to build and equip the present Yavapi Community Hospital opened in 1964. A unique model of what can be accomplished with a full measure of professional medical leadership.

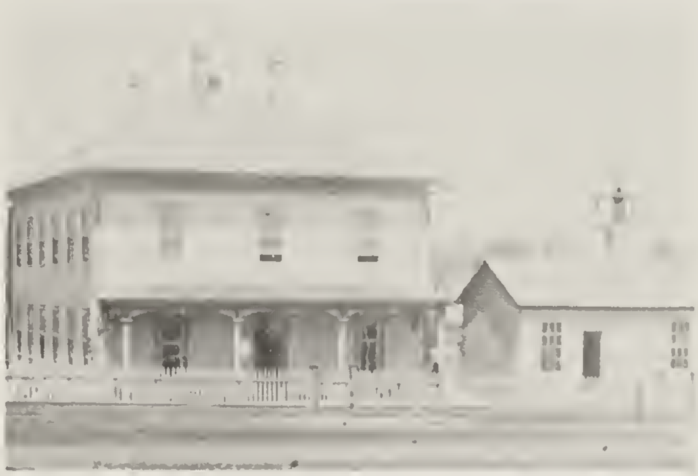
Early Hospitals

Searching for the history of "Sister's Hospital" in Prescott takes us back to 1878, when two nuns of the order of St. Joseph of Carondolet arrived from Tucson for the express purpose of establishing a hospital and a school. They were Mother Superior Mary John Berchman and Sister Monica. Prior to their arrival, civilians with serious illness and injury were admitted to the hospital at Whipple. The Sisters proceeded to organize their program in the Marina-Willis Street area, where the recently arrived Father Beckers had established a chapel.

The arrival of the seven Sisters of this order in Arizona in 1870 is described by Frank Brophy in "Arizona Sketch Book" with liberal quotations from Sister Monica's diary-record of the perilous journey.

The Sisters first rented, then purchased, a two-story frame building, formerly Mrs. Grove's boarding house, on Marina Street, and equipped it for a hospital. The Arizona Miner carried many news items, indicating good community support of their endevors, in the form of balls and benefit theatrical performances, not the least of which was Miss Pauline Markham and her musical troupe presenting "Pinafore". The County Supervisors had a contract waiting for them to care for the indigent ill, and the hospital was soon busy.

In May 1879 there was a threat of excitement as reported in the Enterprise: "Sisters of St. Joseph who have charge of the hospital deserve credit for presence of mind. Fire broke out in the rear of their house. They ran to the spot and began to throw dirt on the powder stored in the burning building, and extinguished the fire and prevented a serious explosion." Why were they storing powder - when it was forbidden by city ordinance? Was it a "service" that the hospital provided for some miner type patient pending his recovery? In those days everyone from the Governor to the chaplain at Whipple had a mine or two out in the hills, but not the Sisters!


Hospital of Sisters of St. Joseph of Carondolet, 1878-1886, on Marina Street.

When Mother Superior Mary John Berchman died in June 1879, her funeral was held at the hospital - a custom of the times - and it was a big one. The editor commented that the only larger ones were those of John Townsend and Gov. Coles Bashford. Pallbearers were prominent business men, officers from Whipple, and Frank Fremont, son of the Governor.

Jessie Benton Fremont, wife of the Governor, had met the Sisters and mentioned them in her correspondence. She had worked with the order in St. Louis during the Civil War, caring for the wounded. She described Sister Monica thus: "All those who indulge in fears and self-pity should see my little St. Monica. Before she came, a mule and a snake frightened her equally. Within a month she was riding alone through the sagebrush and rocks up into the mines, soliciting money for the proposed hospital, and killing snakes with a manzanita cane. Of such stuff Sisters are made."

Scared, or not, Sister Monica became the Mother Superior, and other Sisters came to teach in the school and assist with the nursing. Apparently most of them did both.

For some reason, Mr. H. C. Casey, from whom the Sisters rented, had not been paying his taxes, and in the Yavapai County Recorder's Office we find that a Sheriff's sale was held, and the property was purchased by F. W. Blake for $1617.65 on July 8, 1880. Mr. Blake had a busy assay office, was Wells Fargo Agent, and was Mayor of Prescott, the year the Sisters came. When the required six months after the sale had passed, without any efforts at redemption, Mr. Blake sold the property to the Sisters for $1200. He was not a Catholic, and it is a matter of record that he assisted Methodists, Presbyterians, Congregationalists and Episcopalians in their founding efforts, as well. The whole transaction is meticulously recorded by Sheriff J. R. Walker, himself a member of the famous, historic Walker Party, and a nephew of its leader, Joseph Reddiford Walker. The purchase was made in May 1881, and Sister Monica also paid John G. Campbell $550 for two lots south of the building and Mr. Sullivan for one to the north. In the Recorder's office there is an interesting note on the record of the transaction between Campbell and the Sisters. It said that striking out "heirs" and replacing it with "successors" was done before signing. Improvements were made in the house and grounds, and the newspaper reported a census of 15 patients, one of whom was Dr. Frank Ainsworth.

On September 2, 1881 we read in the Miner: "Mother Monica of Prescott branch of the Sisters of St. Joseph, got back safely, after a trip of seventeen days through the mountains along the grade. She travelled along the grade over 100 miles, visiting every camp, talking kindly, ministering angel as she is, to the workers, who nobly seconded her efforts for forming an association, the object of which is to have sick and wounded people along the road brought here to the hospital, and attend to, as only ladies of her order can attend to suffering mortals. The monthly subscription that will be paid for this purpose, while not heavy on the individual subscribers, will form a large fund, save the county great expense, and be of untold benefit to all who may be so unfortunate as to be in any way injured by accident or prostrated by disease." Blue Cross in 1881!

Father Alfred Quetu, pastor and dynamic builder for Sacred Heart Parish, Prescott, 1889-1908.

In September 1882, Mother Monica was given a new assignment and Mother Justine came to be her replacement. Other changes occured, Bishop Salpointe going to Santa Fe and Father Bourgade becoming Bishop of the diocese. Reports indicate the hospital was still functioning in 1885, but according to a statement in a little parish history (published when the new church was dedicated in 1969) "at the suggestion of Bishop Peter Bourgade in 1886 the hospital was closed and after some remodeling, St. Joseph's Academy was opened in the same little house."

"Hospitals" opened up here and there about town - homes, where some woman with an interest in nursing would take two or three patients. The county hospital, operating in conjunction with the county farm was reported to have 25 patients in 1897, and Dr. Reynolds had a contract for their care for $75 per year. In 1903 there was a grand jury investigation of the county hospital. The findings were that the place was clean and neat, the food palatable and the dining room adequate, but they could find only 26 patients, and not the 44 claimed in reports.

Father Alfred Quetu, a dynamic, able, friendly priest, came to Prescott in 1889, and soon there was much activity about the parish. A beautiful new church was built, and another in Jerome. He was deeply concerned about the sick, and was known to fill up his own house with them. He appealed to the Sisters of Mercy in Phoenix to open a hospital in Prescott. His own sister was a member of the order, supervising art and music in their Academy in Phoenix. In 1893 they came, using a small house near the new church for their hospital.

In 1896 Frank M. Murphy sold to the Mercy Hospital corporation five lots in West Prescott for $750.00, and the City Council voted permission to build a hospital. On January 29 we read in the paper, (it's the Journal-Miner, now) "Work on the new Mercy Hospital will be commenced next week. It is the intention of Father Quetu, who is the prime mover in this enterprise, to erect an imposing structure to cost about $10,000. It is intended for use as a sanitarium to which invalids from abroad will be admitted."

In February excavation for the basement, and contracts for the foundation were let, and then things came to a halt. In November 1896 action resumed, and contracts for material and work, under architect Fred Heinbein, were put in motion. By January 8, 1897, the brick work was nearing completion.

In 1903 when an addition was made, there was a history of the whole project in the paper: "Up to about seven years ago there was no institution in this city, outside of the county hospital and a few private places where the sick could be cared for. At that time a corporation was formed for the purpose of putting up a hospital and the work had proceeded as far as the laying of the foundation when the Sisters of Mercy bought the place and proceeded with the work, in an humble way, building (finishing?) a portion of the building at a time, as the needs of the place increased, and as they had funds to do so, until at last the Mercy Hospital, as it has been the past five years, was completed and equipped. This institution was then adopted for the care of the sick and injured employees of the Santa Fe, Prescott and Phoenix Ry."

HON. FRANK M. MURPHY

Frank M. Murphy, railroad president, mining man, financier and philanthropist. Brother of Gov. N. O. Murphy.

Long time residents report that there was a little frame building behind the brick one that the Sisters operated as a hospital. In the Journal-Miner of May 26, 1897 is this: "The Sisters of Mercy have commenced the removal of their *building* from Marina Street to West Prescott to the site of their new hospital." So it seems it was their plan to continue "business as usual" during the protracted period it was going to take to complete the new facility on Grove Avenue.

Many historical events in the community took place during these years. Electricity came to the area. The Rough Riders were active in the Spanish-American War, costing Prescott its Mayor, "Bucky" O'Neill. In 1900 a great fire destroyed most of the downtown business section, followed by a mammoth re-building. Whipple was being re-built.

The mines were very active. Frank M. Murphy, President of the S. F. P. and P. Ry, was

The first Mercy Hospital begun in 1896, completed and operated by the Sisters of Mercy.

constantly on the move in his private railroad car, bringing famous visitors to the Congress Mine, Castle Hot Springs, or to Prescott. One time, in 1897, he entertained some of them at a garden party at his home on Nob Hill, with music provided by the Whipple Band under the direction of Prof. LaGuardia. (Did son, Fiorello, play cornet that evening?) Murphy was laying out a zoo in West Prescott, piping water and building roads. He gave ten acres to the Sisters of St. Joseph for a new school.

Papers contained ads about shares in gold mines, and impressive testimonials on the value of Lydia Pinkham. The ladies of the Monday Club, recently federated, were building a new library with the help of Andrew Carnegie. A trolly (Prescott and Mt. Union Ry.) was incorporated and laid out. The western terminal would bring passengers to within one long block of the hospital, or they could hike three blocks the other way to the zoo. Automobiles were appearing on the streets and scaring the horses.

The orginal Mercy Hospital soon was crowded and the Sisters recognized: (to continue the 1903 report) "The building would have to be increased in size, and the Sisters went to work to meet the need, and after receiving between $3,000 and $4,000 from private donations, they borrowed $10,000 more and built the new addition which now stands second to no institution in the southwest. There are now about 60 rooms in all the building, and between 50 and 60 patients can be cared for without crowding . . . . The building is three stories high, built of brick with stone foundations . . . Besides the main building there is a smaller building which is used as a ward for the patients having infectious diseases."

Mercy Hospital with 1903 addition. A chapel and convent were built where the water tower stands. This building still exists, the rest has been demolished.

On the opening day of the new addition, the Sisters personally greeted the visitors, guided them around and sent them upstairs for "delightful refreshments."

"The whole institution seems like a parlor with its bright clean walls, polished woodwork, smooth maple floors covered with rich carpets and rugs, elegant curtains and draperies, leather and plush upholstered furniture, mirrors, rocking chairs, etc. all of which is calculated to make things look as cheerful and homelike as it is possible to do."

The medical staff had been enriched by the arrival of some doctors from San Francisco and other cities, including Drs. J. B. McNally, J. S. Barrett, E. W. Dutcher, S. V. Fitzsimmons, H. T. Southworth, J. W. Flinn, C. E. Yount; and Dr. R. N. Looney came in from McCabe. All of these gentlemen had been to Phoenix to take an examination before the newly created Board of Medical Examiners of the Territory.

Another valuable addition was announced in the paper in November of 1903: "The Mercy Hospital in this city was fortunate enough recently to secure the valuable services of Miss Gwendoline Wrenall, a professional nurse of several years experience in some of the best hospitals in the country. She has had considerable experience in line of surgery as assistant to some of the best surgeons in the west and is herself quite proficient in the work, able to perform almost any ordinary surgical operation, and will be a very valuable assistant to the physicians and surgeons of this city in her capacity as nurse at the Mercy Hospital."

There is no record of Miss Wrenall doing surgery, but plenty of evidence that she was an excellent nurse. She was soon joined by another

Jefferson School which became the Prescott Community Hospital in 1943.

trained nurse, Miss Charlotte Walace, who further extended the fine nursing care.

The expanded hospital continued to serve the community until it burned in 1940. Throughout the years, remodeling of the building was done from time to time, the greatest of these being the completion of modern major and minor operating rooms in 1937. The fire was confined to one area, and the patients were evacuated without injury. After the fire the Sisters decided not to rebuild. The furnishings and property were sold, the Sisters assigned to other areas, several going to St. Joseph's Hospital in Phoenix. If the community wished the services of a hospital, it would have to plan one of its own. The era of the Sisters providing one had come to an end.

THE COMMUNITY HOSPITALS

With the Sister's Hospital gone, citizens of Prescott, became active in exploring means of a replacement. The Jefferson Elementary School was vacant because of a shift of school population (and no one had heard of busing). Its rooms and halls were spacious, it was all on one floor, and it was estimated that it would cost $10,000 to alter it into a workable hospital. A public mass meeting was called on December 9, 1940 when it was voted to allow the school board to transfer, by lease, the building, to an association of citizens who would open it as a community hospital. In a test case in February 1941, the State Supreme Court ruled that such a transaction was not legal. The Hospital Association then bought it for $1,000.

An effort was made to get the alterations financed by the WPA, but it was rejected because it was not related to national defense. The Reconstruction Finance Corporation refused a loan because small hospitals were believed to be economically risky. On Oct. 10, 1942, there was a joint meeting of the doctors and the Hospital Association, and it was decided to move at once to alter the Jefferson School into a hospital, relying on local resources for funds.

The war effort was now taking medical and surgical supplies, hospital furnishings, building materials, and man power. Would it be possible to overcome this handicap? Salesmen from surgical supply houses scouted the entire country for needed items. "The last operating table in the U. S." was quickly purchased for a fabulous $1200 by a Yavapai rancher. The doctors also quickly bought needed items. No stoves? An oil-burning commercial kitchen stove was brought in from a defunct mine - courtesy of the bank that held the mortgage. Committees worked to raise cash. Two ranchers had offered $10,000 each, if the townspeople would match the sum. It was done. Some citizens without cash, contributed labor to an effective degree.

Finally on March 1, 1943, the Prescott Community Hospital opened its doors and patients followed the loads of furniture. The first baby was born that evening. On March 20 dedication ceremonies were held, with Governor Sidney Osborn as speaker. He referred to Prescott as a community with internal friction, and hoped that it would become a united one in this hospital effort. History does not permit us to say that the governor got his wish of serenity and peace, for there have been disagreements on various phases of hospital operation. But when the "smoke" clears away, the program moves on.

In the interval between hospitals, Mrs. Catherine Lennox, a registered nurse, had opened a maternity home on South Mt. Vernon Street. She united with the hospital and served as its first Superintendent of Nurses. Staffing the nursing service was difficult. Older nurses came out of retirement, aids were trained, but overtime hours were well known to all.

Community support continued. One group with an outstanding record was the Medical Auxiliary, a small group of ladies, who, between 1952 and 1969 raised $75,000. Lodges, service clubs, and individual "angels" gave generously.

As soon as building materials were available, more rooms were added to the building in 1946, and with help from the Ford Foundation in 1959. A good x-ray machine and services of a radiologist were obtained . The laboratory services were expanded. But with all the stretching, patching and improvising, the needs were outgrowing the building.

The County Hospital, with Hill-Burton funds, replaced their old buildings with a new kitchen in 1952, and two more units in 1955 and 1959. Many citizens came to the conclusion it was economically foolish to duplicate costs, services and staffs, and it was proposed to combine the two operations. On May 2, 1960, a Hospital District (the area of Yavapai County that logically would be served by this hospital) was voted into existance, and a bond issue for $725,000 - to go with $500,000 Hill-Burtons funds - was voted in 1962. The Yavapai Community Hospital opened in 1964, with Prescott Community Hospital patients coming over by ambulance. The new facility had 73 regular beds, 32 beds in extended care, 10 in obstetrics and 16 newborn cribs.

There was a vast improvement in x-ray, laboratory, surgical, and emergency services. Physical and Respiratory Therapy, kidney dialysis were added later, and a very generous gift equipped an intensive - cardiac care unit.

Yavapai Community Hospital Opened in 1964.

Outstanding service has come from the Hospital Auxiliary - the cheerful "Pink Ladies" who have given thousands and thousands of hours of volunteer service, and many gifts of equipment purchased from the profits of their gift shop.

A growing community, a larger medical staff with more specialists, new technology requiring bigger and very expensive equipment for diagnosis and treatment are now making demands impossible to meet in the present plant. On June 1, 1976 a bond issue of 3.1 million dollars was voted favorably to meet the problems. Construction of the additions, and the installation of the new equipment will provide a variety of vastly improved services to the residents of the Yavapai Hospital District.

ACKNOWLEDGEMENTS

Frank Cullen Brophy - "Arizona Sketch Book"
Quebbeman - "Medicine in Territorial Arizona"
C. C. Phillips - "Jessie Benton Fremont: Woman Who Made History"
Sharlot Hall Museum - copies of Arizona Miner, Arizona Journal-Miner, The Enterprise -1878-1905. Also several photos
Yavapai Recorder's Office - Deed Books 13, 14, 34.
Sacred Heart Parishioners: Mrs. Elsie Kapser, Mrs. Vera Keeney, Mrs. Mary Sills, William Vallely, Miss Mamie Duke who supplied personal communications and Miss Emma Andres, who also supplied Sacred Heart Parish Bulletins for 1944.
Don Bell, former Supt. Yavapai County Hospital - Scrapbooks
Yavapai Community Hospital - Scrapbooks. Photos.

JOHN LAWRENCE LE CONTE, M.D.

REV. EDWARD J. PETTID

John Lawrence Le Conte, M.D. was a remarkable man: an eminent American frontiersman of Medicine and of the Natural Sciences; and a pioneer of Arizona and California.

John Lawrence Le Conte was born in New York City on 13 May 1825 to John Eatton Le Conte and Mary Anne H. Lawrence, who died two weeks after her child birth. Among his cousins John Lawrence could name the famous Professors John and Joseph Le Conte of the University of California at Berkeley. Completing his collegiate studies at Mount St. Mary's College, Emmetsburg, Maryland in 1842, John matriculated at the College of Physicians and Surgeons in New York City earning his degree Medicinae Doctoris in 1846. While he avowed Medicine as his vocation, his avocation — Entomology — almost dominated his lifework through a rich inheritance.

John Lawrence adopted this love for Entomology from his renowned naturalist father. This life-long love John nurtured through scientific journeys to mature his knowledge of American Fauna, Geology and Entomology.

When nineteen, and yet a medical student, John published his first scientific paper describing approximately twenty species of Carabidae

Very sincerely yours
John L. LeConte

From Mercy Hospital & Medical Center, 4077 Fifth Ave., San Diego, CA 92103.

from the eastern United States. Early in his career he correlated several species of Coleoptera common to North America and Europe. he solidified this work in his essay on the distribution of California beetles read to the American Association in 1851. In 1860 he surveyed more elaborately and extensively the West in his "Coleoptera of Kansas and New Mexico".

In quest for his "El Dorado" of elusive beetles and other scientific phenomena, Dr. Le Conte sailed by sidewheeler from New York with a December 1849 stopover on the Spanish Main's Isthmus of Panama before proceeding past Acapulco, San Blas, Mazatlan, the pearl beds of the Sea of Cortes and through the Golden Gate of California.

After exploring the San Francisco Bay area, then during March the Santa Clara Valley and southern waypoints for beetles, he followed the dusty coastal stage coach road to San Diego.

There John startled the local gentry by toeing into and sifting the seashore sand; kicking aside the broad, brown, slimy leaves and broken necklaces of dripping seaweed on the then ecologically-clean beaches of San Diego and Coronado. From New Town north past Old Town, in the salt marshes too of False Bay, constantly he searched for unknown species of beetles.

John reconnoitered some twelve miles north of San Diego. Later he alerted Dr. C. C. Parry of the U.S. Boundary Commission to a peculiar species of native pine with a dense cone and long, stout leaves, five in a sheath. This rare, wind-blown pine grew on the high, dark reddish and tan-colored sandstone cliffs overlooking the Pacific Ocean. These gnarled conifers struggled to survive amid the coastal sage-scrub and high chaparral: California buckwheat, prickly pear, lemonadeberry, red monkey flowers, chamise and black sage. These soft-soughing Torrey pines sheltered the quick quail and the twitchy-nosed rabbit. Doctors Le Conte and Parry named these Torrey pines honoring their mutual New York City mentor and friend, Dr. John Torrey.

During this time the United States Second Infantry was camping up Mission Valley a few miles east of the beach near the ruined and relocated Mission San Diego de Alcala. Brevet Major Samuel Peter Heintzelman commanded them.

At least by Thursday, the 8th day of October in 1850, Dr. Le Conte met the Brevet Major and dined with him and some regimental officers in San Diego. There and then he doubtless encountered Captain Delozier Davidson, 1st Lieutenants Ed. Murray, Henry B. Hendershott, and 2nd Lieutenant Thomas W. Sweeny. He also happily renewed friendship with Assistant Surgeon Henry Stuart Hewit: a Class of '47 fellow alumnus of the New York College of Physicians and Surgeons. He greeted too the soldiers of Companies D, H. and I, 2nd Infantry who, on 23 October, would take up the line of march following the San Diego River up to Santa Ysabel near the summit of the Coast Range and down and across the desert to the confluence of the Colorado and Gila Rivers (later Fort Yuma) to protect the "Forty-Niners" and other American emigrants from the villainous Whites and hostile Redskins alike.

Before departing San Diego, Dr. Le Conte and some Army officers invested in land in the newly-surveyed "New Town". Major Heintzelman bought eighteen lots; Lt. Murray, one; 2nd Lt. Sweeny, two. Dr. John bought two lots from Don Jose A. Aguirre on 18 September 1850: one was located on the present Navy athletic field at the foot of India Street; the other lay opposite the old Plaza de Pantoja on State Street bayside.

That same late Autumn of 1850 rumors floated around San Diego about a mysterious active volcano in the middle of a salt lake somewhere east of Santa Ysabel. Major Heintzelman decided to investigate He invited Dr. Le Conte to accompany him as a scientific observer to this strange desert land north of the white shifting sand dunes.

Early on the morning of the 28th of October this small expedition took off from San Felipe guided by a Santa Ysabel Mission Indian and the Digueno Indian Chief who lived not far from this "volcano". After following a general north-easterly heading over precipitous metamorphic rocky terrain scantily clad with prickly pear cactus, greasewood, ocotillo, mesquite, smoke trees, and other desert vegetation, by 8:00 a.m. that same morning they scanned an immense plain broken by craggy mountain ranges and serpentine arroyos stretching distantly to the eastern horizon. A mirage-like dry salt lake shimmered as a silver streak framed by a frosty-white shoreline and misted over by a wispy, whimsical fog. They needed no great imagination to envision a future over-swollen Colorado River discharging its surplus floodwaters into this vast below-sea-level desert sink measuring about ten by thirty miles to form a "Dead Sea".

Remounting their horses they continued eastward until encamping about 4:00 p.m. beside salty Carriso Creek. After riding for seven hours the following day, they approached an Indian village on New River, near or inside, the present Salton Sea where the local Indians annually scraped together their salt supply.

Further another eight miles, they reached an elevated, some 100 feet, chain of eight volcanic mounds of lava and pumice. Tall, stately plumes of cottonish-white columns of steam blew and squealed from bubbling conic mud holes which exhaled also a hellish sulphurous stench. Four blow holes violently jetted live steam and shot boiling saline water high into the air. Another hot spring ebbed and overflowed with foaming brine a ranbow-colored, salt-encrusted basin.

Closing on these smoking springs as cautiously and softly as stalking cats, the reluctant Indian guides then stalled in their tracks and tried to persuade the Americans from approaching more closely. The nervous Diguenos explained that, according to their beliefs, whoever brashly adventured too closely would be killed by dirty devils grotesquely misshapened like huge black birds who would flap heavily into the witchy air and crashdive into them.

The Americans were quite impressed with this bit of Indian folklore but they kept coming — cautiously, step by feathery step until they stood in the middle of this land of a hundred smokes.

Seeing that the flying balck devils did not destroy the bold and likeable Americans, the happy Indians unrestrainedly jumping and dancing for sheer joy ran toward the Americans whose smiles of achievement quickly turned to instant wrinkles of concern. For the highly-excited Indians shook the thin, trembling earth crust so terribly that they nearly broke through the crust-cover which would have dropped everyone into a finely-vaporizing, high-pressure steam cauldron.

Finishing their scientific observations, the American soldiers and Indians horsebacked several days to Vallecitos to rejoin the main body of troops awaiting since 3 November to resume marching to the Colorado River.

From embryonic Fort Yuma, Dr. Le Conte launched out to investigate the Colorado River, this Buena Guia or Rio del Tizon, with Doctor George Ogden of Mississippi, President of the Colorado Ferry Company. In an open skiff these two men drifted while measuring and exploring this giant river downstream one hundred miles till encountering brackish tidewater. While the river's depth averaged nine feet, the shallowest depth of the channel, five feet, was fourteen miles below the Gila's mouth. The Colorado meandered and the current coursed quickly as the waters deltaed into the Gulf of California. Alongside both banks eddied a few half-sunken small trees unlikely to sang river traffic. But Le Conte observed an oddity: as the mud red waters neared the Gulf, in some places it did not gouge out the lowest level of land. But due to continual silt and sediment depositing to form parallel levees like an aqueduct, the riverwaters ran channelized above the surrounding desert to the sea.

This voyage of rediscovery—for its mouth had been previously discovered by Ulloa in 1539; navigated possibly up to the Gila junction by Alarcon in 1540; forded by melchior Diaz in that same year—confirmed an avenue of water communication into the heart of the desert and assured that steamboats could churn up and down the Big Red. The actual soundings of Le Conte and Ogden proved that shallow draft freighters could safely steampaddle from the Pacific Ocean via the Gulf of California and, after threading between Tiburon and Angel de la Guarda Islands, enter the Colorado River to discharge their passengers and cargo at Yuma.

After their return to Fort Yuma, Dr. Ogden chatted with Major Heintzelman about the findings about the Colorado River, and, during the conversation, said that Dr. Le Conte planned a trip into Sonora with a Mexican guide to purchase forage, transport and sell it to the U.S. Government at Fort Yuma. Dr. John hoped to make a small fortune because the price was quite inflated due to the great southwestern drought and the destruction of Yuma Indian food supplies by General Morehead the summer before.

Armed with this information, the Major was fortified for Dr. Le Conte's call at the Commanding Officer's tent on Friday, 10 January 1851. The Doctor first requested a passport. Then he abruptly changed his request to a letter of recommendation. Jarred by this quick change, the Major became doubly suspicious especially when the Doctor further explained that the principal purpose for the proposed trip was pure Science.

To be continued

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

ARTICLES OF INCORPORATION AND BYLAWS COMMITTEE

The meeting of the Articles of Incorporation and Bylaws Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona on August 7, 1976, convened at 2:02 p.m., Charles E. Henderson, M.D., Chairman presiding.

MINUTES

The minutes of the meeting held February 20, 1976 were approved as distributed.

RESOLUTION 30-76

RESOLVED, That Chapter VIII, Section 6 be amended to read:

Section 6. Reference Committees: — At or before each Annual Meeting, the Speaker shall appoint the following reference committees from among the Delegates: Credentials; Nominations; Reports; Amendments; Resolutions; and such other committees as the Speaker may from time to time designate. Each of such committees, unless otherwise specified, shall consist of at least three members, who shall serve during the Annual Meeting for which they are appointed.

(a) The Committee on Credentials shall examine the credentials of each Delegate or alternate claiming membership in the House, and recommend the seating of those holding proper credentials. It shall also report to the Speaker as to whether a quorum is present and its members shall act as sergeants at arms.

(b) The Committee on Nominations shall be a committee of at least three selected from and representing the Nominating Committee, as described in Chapter V, Section 5, shall present the nominations of the Nominating Committee to the House, and shall act as Tellers.

(c) The Committee on Reports shall receive and review the reports of officers, the Board, and standing and special committees in advance of the first session, and shall report thereon at the first session.

(d) The Committee on Amendments shall receive all proposals to amend the Articles of Incorporation and these Bylaws, presented and read at the first session; and the time and place of the committee meeting shall be posted and announced in order that proponents and opponents of proposed amendments may be heard. The committee shall make its report to the House at the second session.

(e) The Committee(S) on Resolutions shall receive and review all resolutions and other proposals not specifically delegated to another reference committee, introduced at the first session. The committee shall consider the same at a meeting, the time and place of which shall be posted and announced in order that proponents and opponents of the various resolutions and proposals may be heard. The committee shall make its report thereon at the second session of the House. New business, not provided for in the orders of the day for the second session, cannot be considered at that session except by suspension of the rules.

RESOLUTIONS MAY BE PROPOSED BY (A) THE BOARD, (B) ANY DELEGATE, (C) ANY COUNTY SOCIETY, (D) ANY COMMITTEE OF THE ASSOCIATION, OR (E) BY A PETITION SIGNED BY TWENTY OR MORE MEMBERS OF THE ASSOCIATION.

AT THE DISCRETION OF THE SPEAKER, MORE THAN ONE COMMITTEE ON RESOLUTIONS MAY BE APPOINTED IN ACCORDANCE WITH CHAPTER VIII, SECTION 6. REFERENCE COMMITTEES.

NOTE: All bracketed, italicized words represent deletions; all fully capitalized words are new material.

UNIFIED MEMBERSHIP

It was noted that a membership poll is currently underway regarding the continuation of unified membership with the American Medical Association. Should the poll results be such that the Board of Directors might want to hold a special session of the House of Delegates this committee prepared the following Resolution for use by the Board of Directors should they desire to use same.

RESOLVED, That the Arizona Medical Association make the following amendments to its bylaws:

CHAPTER II — Membership

Section 1. General Requirements, be amended to read:

An active member in good standing of a county society is automatically an active member of this Association, and, conversely, an active member in good standing of this Association must be an active member of a county society.

Any person, when he becomes a member, shall agree to abide by the Articles of Incorporation and Bylaws of this Association, and by any lawful changes which from time to time may be made. He further agrees to abide by the Code of Ethics as laid down by the American Medical Association as it now exists, or may hereafter be amended, *(and, be a member of that association.)*

Section 4. Loss of Membership, be amended to read:

(A) Active, Associate or Fifty-Year Club membership in this Association shall be lost by:

(i) Final expulsion from the county society through which he is an active member of the Association.
(ii) Transfer of membership to a county society of another state, and thereby to another state association, except as provided in Chapter III, Section 3.
(iii) Voluntary resignation, which shall be without prejudice.
(iv) Action of the House of Delegates (see Chapter VI, Section 6; Chapter VIII, Section 9).
(v) Revocation of the member's license to practice in Arizona. This later clause shall effect immediate loss of membership as of the date of revocation.
(vi) Termination of Board of Medical Examiners registration of intern or resident active member. This latter clause shall take effect at the end of the calendar year.
((vii) Failure to maintain membership in good standing in the American Medical Association.)
(vii) Failure to pay Association dues and assessments within one year of the date such become payable, unless such failure be exempted under Chapter IX, Section 5.
(viii) For active membership, failure to fulfill the continuing medical education requirements of the Arizona Medical Association elsewhere in the bylaws.
(B) Active, Associate or Fifty-Year Club membership in this Association shall be suspended by:
(i) Suspension of membership in the county society through which he is an active member of this Association.
(ii) Action of the Board (see Chapter VI, Section 6).
((iii) Suspension of membership in the American Medical Association, or)
(iii) Failure to pay the annual dues and assessments before the delinquency date.
(iv) For active membership, failure to submit satisfactory reports showing compliance with the continuing medical education requirements of the Arizona Medical Association.
All of the rights and privileges of membership shall be withheld during the period of suspension of membership.

(C) Service, honorary, student, and affiliate membership in this Association may be withdrawn upon a majority vote of the House after recommendation of the Board.

(D) The county society involved, and the Association, shall each notify the other of any change of status under these provisions for loss or suspension of membership.

(E) Any physician who may feel aggrieved by action of the society of the county in which he resides in refusing him membership, or in suspending, censuring, or expelling him, shall have the right to appeal to the Board, and IF HE OR SHE IS A MEMBER OF THE AMERICAN MEDICAL ASSOCIATION AND, if he so desires, further to the Judicial Council of the American Medical Association.

Any physician suspended, or censured, by the Board, or expelled by action of the House, or any county society, which is a party to such appeal to the Board, may appeal such of these decisions, as the Judicial Council of the American Medical Association will accept, to the Judicial Council for review. In this event, the decision of the Judicial Council will be final. In the event the Judicial Council refuses to consider such an appeal, the decision of the Board, or the House, as the case may be, shall be final. In hearing appeals, the Board may admit such oral or written evidence as in its judgment will most fairly present the facts, but in the case of every appeal the Board as a Board and as individuals shall first make effort at conciliation and compromise.

CHAPTER V — Election of Officers and Members of the Board Section 1. General: Qualifications: — All elections for officers shall be conducted as a part of the business of the regular annual meeting of the House; shall be by secret, written ballot, and the candidate receiving a majority vote for a particular office shall be elected to that office. If no one of three or more candidates for a particular office shall receive a majority of the votes cast, the two with the highest number of votes shall be the candidates in a run-off election. If there are two candidates only, and the vote is a tie, there shall be a run-off election. No person shall be eligible for election to STATE office, or to the Board, who has not been a member of the Association for the preceding three years, and who was not a Delegate or Director at the time of his election. NO PERSON SHALL BE ELIGIBLE FOR NOMINATION AS DELEGATE OR ALTERNATE DELEGATE TO THE AMERICAN MEDICAL ASSOCIATION FOR THE PRECEDING THREE YEARS, AND WHO WAS NOT A STATE DELEGATE OR DIRECTOR AT THE TIME OF HIS ELECTION. All officers and members of the Board shall serve until their successors are elected and have accepted the office.

Section 3. Terms of Office, Qualifications, be amended to read:

The following officers shall be elected for a one year term: the President-elect, Vice President, Secretary, Treasurer, Speaker of the House, and Editor-in-Chief. The President and Past-President, though not elected as such, also have a one year term.

The Delegate(s) and Alternate Delegate(s) to the American Medical Association shall be elected in accordance with the Bylaws of the American Medical Association. If the Association is entitled to more than one Delegate, not all terms shall run concurrently. ANY NOMINEE FOR DELEGATE(S) OR ALTERNATE DELEGATE(S) TO THE AMERICAN MEDICAL ASSOCIATION SHALL ALSO BE A MEMBER OF THE AMERICAN MEDICAL ASSOCIATION.

District Directors shall be elected for a term of three years.

ONLY THOSE MEMBERS OF THE HOUSE OF DELEGATES WHO ARE MEMBERS IN GOOD STANDING WITH THE AMERICAN MEDICAL ASSOCIATION SHALL BE ELIGIBLE TO VOTE FOR DELEGATES AND ALTERNATE DELEGATES TO THE AMERICAN MEDICAL ASSOCIATION.

CHAPTER IX — Dues and Assessments

Section I. Fixing of Annual Dues; Payments; Reinstatements; Collections; Enforcement; be amended to read:

(a) The amount of annual dues shall be determined by the House at the Annual Meeting and shall include a subscription to the Journal. Any proposal to increase the annual dues shall be submitted to the Delegates at least six weeks prior to the Annual Meeting.

(b) The annual dues and the dues and assessments of the American Medical Association shall be payable January first of the year for which levied and shall be delinquent after February fifteenth of that year. The secretary of each county society shall collect and forward to the Association Secretary, the dues for its members, together with the dues and assessments levied by the American Medical Association, FOR THOSE PHYSICIANS WHO ARE ITS MEMBERS.

(c) Any member whose dues for the current year, *(or whose dues and assessments for the American Medical Association)* have not been remitted to the Association Secretary on or before February fifteenth, shall stand suspended until such delinquency is corrected.

(d) The Board may, upon the recommendation of a county society, authorize installment payment of dues by a member. The first payment shall be delinquent after February 15 of the year for which such dues are levied. The delinquent dates for subsequent payments shall be determined by the Board but in any event the final payment would be delinquent after October 1. Any member whose payment has not been remitted to the Association Secretary on or before the delinquent date shall stand suspended until such delinquency is corrected. Any installment program for paying dues and assessments for the American Medical Association shall be determined by the American Medical Association.

Note: All bracketed, italicized words represent deletions; all fully capitalized words are new material.

MECHANICS OF VOTING

Mr. Robinson was directed to investigate methods of voting when only a portion of the House of Delegates are members of the American Medical Association.

Meeting adjourned 3:08 p.m.

William E. Crisp, M.D.
by
Bruce E. Robinson
Executive Director

SCIENTIFIC ASSEMBLY COMMITTEE

The meeting of the Scientific Assembly Committee of the Arizona Medical Association, Inc., held Saturday, August 21, 1976, at 810 W. Bethany Home Road, Phoenix, Arizona convened at 1:07 p. m., Luis S. Tan, M. D.

MINUTES

The minutes of the meeting held December 13, 1975 were approved as distributed.

VICE-CHAIRMAN

Richard A. Silver, M. D. was unanimously elected as vice-chairman with the understanding that he will be the chairman for the 1978 meeting to be held in Tucson.

WEDNESDAY NIGHT STEAK FRY

Discussion ensued regarding the cost of the Annual Steak Fry resulting in the following actions:

IT WAS MOVED AND CARRIED TO PROVIDE ONLY TWO FREE TICKETS TO EACH FIRM PURCHASING AN EXHIBIT SPACE.

IT WAS MOVED AND CARRIED TO ELIMINATE THE OPEN BAR POLICY BUT TO PROVIDE TWO FREE DRINKS WITH EACH TICKET TO THE STEAK FRY. THIS POLICY TO APPLY TO COMPLIMENTARY TICKETS AS WELL AS TO TICKETS THAT ARE PURCHASED.

FUTURE MEETINGS

Mr. Robinson reported that the 1977 meeting has been confirmed for April 26-30, 1977 at the Phoenix Hyatt Regency.

That the 1978 meeting has been confirmed for April 25-29, 1978 at the Marriot (formerly the Braniff Place) Hotel and the Tucson Convention Center in Tucson.

1979 Meeting Location and Dates

Howard Hyde, M. D., made a presentation to the committee regarding holding a future meeting in Casa Grande. it was pointed out that with the present format wherein approximately 12,000 square feet are needed for exhibit space, neither of the hotels in Casa Grande could accommodate our meeting.

IT WAS MOVED AND CARRIED TO AUTHORIZE MR. ROBINSON TO MAKE TENTATIVE COMMITMENTS WITH THE SAFARI HOTEL IN SCOTTSDALE AND THE HYATT REGENCY IN PHOENIX FOR THE 1979 MEETING SCHEDULED FOR APRIL 24-28, 1979. FINAL DECISION TO BE MADE AFTER THE 1977 MEETING.

1978 School

IT WAS MOVED AND CARRIED TO INVITE THE FOLLOWING SCHOOLS, IN THE ORDER LISTED, TO PROVIDE THE FACULTY FOR THE 1978 MEETING.

1. UNIVERSITY OF CALIFORNIA, IRVINE-CALIFORNIA COLLEGE OF MEDICINE
2. STANFORD UNIVERSITY SCHOOL OF MEDICINE
3. UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

NEW FORMAT

A new format for the 1977 meeting along the following lines was discussed at great length.

Tuesday - 6 am - Executive Committee
9 am - Board of Directors
1 pm - House of Delegates

Wednesday - 7 am to 1 pm - Scientific Presentations
1 pm - Specialty Luncheons
2 pm - Reference Committees
7 pm - Steak Fry

Thursday - 7 am to 1 pm - Scientific Presentations
1 pm - Specialty Luncheons
1 pm - Golf & Tennis
7 pm - ArMPAC

Friday - 7 am to 1 pm - Scientific Presentations
1 pm - Specialty Luncheons
Balance of Afternoon free
7 pm - President's Banquet

Saturday - 8 am - House of Delegates

IT WAS MOVED AND CARRIED TO NOT ADOPT THE SUGGESTED NEW FORMAT AND TO RETAIN THE SAME GENERAL SCHEDULE AS HAS BEEN UTILIZED DURING THE PAST SEVERAL YEARS.

POST GRADUATE COURSES

IT WAS MOVED AND CARRIED THAT THERE WOULD BE TWO IN DEPTH POST-GRADUATE COURSES OFFERED DURING THE 1977 MEETING. EACH COURSE WOULD RUN FOR EIGHT HOURS ON THE FOLLOWING SCHEDULE: 7 AM TO 12 PM TO 5 PM WITH ONE SUBJECT ON THURSDAY AND THE SECOND SUBJECT ON FRIDAY. THAT THERE WOULD BE A $25.00 FEE. THAT ADVANCE REGISTRATION WOULD BE REQUIRED WITH THE DEADLINE FOR REGISTRATION TO BE TWO MONTHS PRIOR TO THE ANNUAL MEETING. THAT WE WOULD REDUCE THE NUMBER OF ROOMS USED FOR THE REGULAR PROGRAM TO THREE SO THAT THESE PARTICULAR COURSES WOULD RUN CONCURRENTLY WITH THE REST OF THE PROGRAM. REGISTRATION TO THESE SPECIAL COURSES WOULD BE DETERMINED BY THE ROOM SIZE.

THE TWO SUBJECTS WOULD BE:

TRANSACTIONAL ANALYSIS
SPEAKER: Dr. Tan to discuss with the U of W

INFECTIOUS DISEASE
SPEAKER: George Ray, M. D.

NOTE:

SHOULD THE U OF W NOT BE ABLE TO PROVIDE AN APPROPRIATE PERSON TO CONDUCT THE COURSE ON TRANSACTIONAL ANALYSIS, AN ALTERNATE TOPIC WOULD BE PAIN WITH DR. TAN TO DISCUSS THE CHOICE OF SPEAKER WITH THE U OF W.

SPECIAL PROMOTION WOULD BE GIVEN TO THESE COURSES.

AUXILIARY

IT WAS MOVED AND CARRIED TO HAVE ONE SPECIAL PROGRAM WHICH WOULD BE PREPARED BY THE AUXILIARY. THIS PROGRAM WOULD BE HELD ON THURSDAY FROM 3:30 PM TO 5 PM, THE SUBJECT WOULD BE: UNDERSTANDING THE TEENAGER, SPEAKERS TO INCLUDE PEDIATRICIANS GEORGE D. COMERCI, M. D. AND JANE SCHALLER, M. D. PLUS AN ENDOCRINOLOGIST YET TO BE NAMED.

Auxiliary Tentative Program

Mrs. Hoffman presented the following tentative program.

Wednesday, April 27

9-4 Registration
9 - 4 Hospitality Center
9 - 4 Arts & Crafts Show
9 - 4 AMA - ERF Booth

11 to 12 N PRE—CONVENTION BOARD MEETING
Brunch (Breakfast Meeting)
State Officers and Chairmen and
County Presidents in attendance

MINI-WORKSHOP

12:30 - 1:45 pm MULTI-COMMITTEE MEETINGS (Luncheon)

GAVEL CLUB
County Presidents (In & Out going)
Newsletter Editors
Hammer Loan Fund
Legislation *(may decide upon lunch at the capital)
Treasurers

7 - 8 pm Annual Reception
8 pm Annual Steak Fry

Thursday, April 28

9 - 4 Registration
9 - 4 Hospitality Center
9 - 4 Arts & Craft Show
9 - 4 AMA-ERF Booth

9 am to 10 am BREAKFAST FOR IN & OUT GOING ('76-77)
State officers, chairmen, and county presidents (also component Maricopa county auxiliary presidents) purpose: turn over books and materials and discuss duties and ideas

10:15 am - 11:30 am FIRST GENERAL SESSION
All auxiliary members invited included in agenda:

ELECTION OF OFFICERS

12 Noon Luncheon with entertaining speaker or program

3:30 to 5 pm UNDERSTANDING THE TEENAGER (panel)

Tennis tournament
Bridge tournament
A tour of historic house in Phoenix
Shopping trip planned to quaint stores

7 - 8 ArMPAC Annual Reception
8 ArMPAC Annual Dinner

Friday, April 29

9 - 12 Noon Registration
9 - 12 Noon Hospitality Center
9 - 12 Noon Arts & Crafts Show
9 - 12 Noon AMA - ERF Booth
9:30 - 11:30 SECOND GENERAL SESSION
All auxiliary members invited
Memorial Service
AMA Auxiliary President Report
ArMA Auxiliary President's Report

12 Noon LUNCHEON/FASHION SHOW
ArMA President's Address: Dr. Kahle
Greetings from AMA Auxiliary President
ArMA President's Report
Installation of Officers
Pin & Gavel presentation to new President
Acceptance Speech by Maude Reno
Adjournment
Fashion Show

*****Immediately following the luncheon there will be:**
POST CONVENTION BOARD MEETING
1977-78 ArMA Auxiliary Board in Attendance

FREE AFTERNOON

6:30 pm - 7 pm ArMA Auxiliary President's Reception (by invitation only)
7:00 pm - 8 pm ArMA President's Reception
8 pm President's Banquet

SURVEY

Dr. Tan pointed out that he caused to have letters written to specialty societies, committee members, moderators and others soliciting suggestions and comments for the 1977 meeting. Responses were received from the following and were reviewed by the Committee:

A. Arizona Society of Internal Medicine
B. Arizona Academy of Family Physicians
C. Arizona Chapter - American Academy of Pediatrics
D. Robert C. Cannell, M. D., letter 5/24/76
E. Thomas E. Bittker, M. D., letter 5/24/76
F. Tucson Psychiatric Society
G. Philip Levy, M. D., letter 5/13/76
H. William A. McGary, M. D., letter 5/20/76

UNIVERSITY OF WASHINGTON FACULTY

The committee reviewed the letters dated 7/8/76 and 7/21/76 from John N. Lein, M. D., Associate Dean, University of Washington School of Medicine who is our liaison for the 1977 program. Following extensive discussion it was determined to invite the following speakers to present the topics as listed:

PSYCHIATRY

Speaker: Carl Eisdorfer, M. D. *OR* Murray Raskind, M. D. - Dr. Tan to decide who

Topics:

1. PSYCHOPHARMACOLOGY FOR THE ELDERLY
2. ALCOHOLISM - Dr. Tan to discuss this topic with the U of W as it was not on the original list of topics presented by the U of W.
3. Dr. Tan is to discuss the combining of the following three topics into one, and to arrive at an appropriate title.
   a. SENILE DEMENTIAS - LATEST FINDINGS IN IDEOLOGY, DIAGNOSIS, AND TREATMENT.
   b. TREATMENT OF BEHAVIORAL DISTURBANCES IN OLDER PERSONS IN THE COMMUNITY.
   c. TREATMENT OF BEHAVIORAL DISTURBANCES IN THE AGED IN LONGTERM CARE FACILITIES.

Speaker: David Raskind, M. D.

Topics:

1. SUICIDE
2. TREATMENT OF INSOMNIA
3. Dr. Tan is to discuss combining the following three topics into one, and arrive at an appropriate title.
   a. TTREATMENT OF DEPRESSION
   b. MEDICAL ASPECTS OF DEPRESSION

INTERNAL MEDICINE

Speaker: Alex Fefer, M. D.

Topics:

1. CURRENT CONCEPTS OF IMMUNOLOGY
2. THE IMMUNOLOGICAL BASIS FOR CANCER
3. CHEMOTHERAPY IN THE TREATMENT OF CANCER

Speaker: Marvin Turck, M. D.

Topics:

1. ANTIBIOTIC THERAPY
2. URINARY TRACT INFECTION - Dr. Tan to discuss this with the U of W as it was not on the original list of subjects offered.
3. Dr. Tan to discuss the subject of the third presentation with the U of W.

SURGERY

Speaker: James Carrico, M. D.

Topics:

1. CURRENT CONCEPTS IN THE TREATMENT OF SHOCK
2. INITIAL EVALUATION OF A TRAUMATIZED PATIENT
3. SYSTEMS FOR MONITORING THE SEVERELY INJURED PATIENT

Speaker: Michael K. Copass II, M. D.

Topics:

1. THE COMATOSE PATIENT
2. SPINAL CORD INJURIES
3. THE TREATMENT OF DRUG OVERDOSES.

SPORTS INJURIES

Speaker: James Garrick, M. D.

Topics:

1. THE FEMALE ATHLETE, A SPECIAL PROBLEM
2. Dr. Tan to discuss the idea of Dr. Garrick conducting a program from 2 PM thru 5 PM (with half hour break between 3 PM and 3:30 PM) on Friday afternoon to which coaches, trainers, physical education teachers as well as physicians would be invited. Richard A. Silver, M. D. has volunteered to moderate this presentation. Such a presentation to cover the following subjects as well as to provide ample time for questions and discussion from the floor.
   a. RECOGNITION AND SIDELINE MANAGEMENT OF HEAD AND NECK INJURIES
   b. CHOOSING, FITTING AND MAINTENANCE OF PROTECTIVE EQUIPMENT
   c. THE ATHLETES' ANKLE
   d. TREATMENT OF SKI INJURIES
   e. THE USE AND ABUSE OF PROTECTIVE

EQUIPMENT IN SPORTS

f. NUTRITION IN ATHLETES

PEDIATRICS

Speaker: Jane Schaller, M. D.

Topics:

1. RHEUMATOLOGY
2. Dr. Schaller would participate in the Auxiliary panel, UNDERSTANDING THE TEENAGER scheduled for Thursday from 3:30 pm to 5 pm. Dr. Tan to discuss this with the U of W as it was not on the original list of topics offered.
3. Dr. Tan to discuss the subject of the third presentation with the U of W.

Speaker: Vincent C. Kelly, M. D.

Topics:

1. ENDOCRINOLOGY
2. Dr. Tan to discuss the subject of the second presentation with the U of W.
3. Dr. Tan to discuss the subject of the third presentation with the U of W.

OB-GYN

Speaker: Leon Spadoni, M. D.

Topics:

1.
2. Dr. Tan to ask Dr. Spadoni to choose three topics.
3.

Speaker: Kent Ueland, M. D.

Topics:

1. HIGH RISK PREGNANCY
2. DIABETIC PREGNANCY - Dr. Tan to discuss with U of W as topic was not on the original list offered.
3. Dr. Tan to discuss the subject of the third presentation with the U of W.

GENERAL

Speaker: John N. Lein, M. D.

Topics:

1. CONTINUING MEDICAL EDUCATION - This is to be a breakfast presentation which may include others if Dr. Lein so chooses. Dr. Tan to discuss with Dr. Lein as it was not on original list offered.
2. Dr. Tan to discuss with Dr. Lein the subject of the second presentation.
3. Dr. Tan to discuss with Dr. Lein the subject of the third presentation.

BREAKFAST PANELS

It was determined to have two breakfast panels as follows:

Thursday - 7:30 am to 8:45 am

Topic: CONTINUING MEDICAL EDUCATION

Friday - 7:30 am to 8:45 am

Topic: PHYSICAL FITNESS FOR THE PHYSICIAN

Speaker: Mr. Robinson to investigate the *PHYSICAL FITNESS INSTITUTE OF AMERICA* as possible presentors. Dr. Tan to make final decision if this organization not appropriate.

CORRESPONDENCE

Claude A. Frazier, M. D. letter 6/14/76

It was determined to thank Dr. Frazier for his offer to participate in our 1977 meeting, however we will be unable to accept.

OTHER BUSINESS

Financial Report for 1976 Meeting

Mr. Robinson presented the subject report as follows:

Revenue		
441	Exhibit booth rental	$14,500.00
442	Sponsorship, speakers	2,800.00
443	Steak Fry	5,489.00
444	President's Banquet	3,877.00
445	Breakfast Panel #1	736.25
446	Breakfast Panel #2	769.75
447	Registration Fees	18,430.00
448	B S Share of Expense	8,000.00
449	Other	-0-
	Total Revenue	$54,602.00
Expenditures		
541	Exhibit booth expense	1,477.60
542	Guest speaker expense	7,114.62
543	Steak Fry	7,436.45
544	President's Banquet	7,215.30
545	Breakfast Panel #1	726.00
546	Breadfast Panel #2	924.00
547	Audio-visual equipment	2,703.17
548	Delegate's Manual	4,542.87
549	Employee hotel	2,426.97
550	Gratuities, hotel emp.	225.00
551	Miscellaneous	2,966.90
552	Newcomer Program	40.00
553	Nominating Committee	36.26
554	Postage	-0-
555	Printing	3,954.10
556	Promotion	3,979.31
557	Public Relations	2,118.63
558	Scientific Assembly Committee	1,116.74
559	Supplies	1,351.74
560	Travel	28.80
561	Telephone	591.03
	Total Expenditures	$50,975.49
		$ 3,626.51

Specific Scheduling

It was agreed that the chairman would be responsible for scheduling specific speakers to specific time periods.

Next Meeting

The next meeting will be held as soon after the first of December as possible for the purpose of reviewing local scientific paper applications and scientific exhibit applications which will be solicited during the month of September.

Meeting adjourned 5:28 p. m.

William E. Crisp, M. D.
Secretary
by
Bruce E. Robinson
Executive Director

EXECUTIVE COMMITTEE

The Executive Committee meeting of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, AZ on Friday, August 27, 1976 convened at 7:04 p.m., Edward Sattenspiel, M.D., President and Chairman, presiding.

MINUTES

The minutes of the meeting held June 11, 1976 were approved as distributed.

MICA-ArMA MODIFYING AGREEMENT

IT WAS MOVED AND CARRIED TO APPROVE THE SECOND MODIFYING AGREEMENT AS FOLLOWS:

SECOND MODIFYING AGREEMENT

Date: As of August 9, 1976,

Parties: ARIZONA MEDICAL ASSOCIATION, INC., a corporation herein called ArMA, and MUTUAL INSURANCE COMPANY OF ARIZONA, a corporation herein called MICA, **A G R E E D:**

Recitals:

1. **Agreement for Payment of Organization Expenses.** As of February 24, 1976, the parties entered into a certain FINANCING AND SERVICE AGREEMENT whereby, in Paragraph 3.A., ArMA agreed to pay and discharge, as they might accrue, (1) all expenses which may have heretofore accrued in connection with the organization and incorporation of MICA, (2) all expenses of MICA connected with its application for and the issuance to it of a Certificate of Authority authorizing it to carry on and conduct a professional liability (malpractice) insurance business in the State of Arizona, (3) all expenses of MICA in connection with the filing by it of an application for the registration of securities and the issuance to it of a permit authorizing the sale of securities in the State of Arizona pursuant to the Securities Act of Arizona, (4) all expenses which may be incurred by MICA in connection with any application for and issuance of a license as a dealer in its own securities, (5) all printing expenses in connection with the printing of any prospectus or offering circular necessary to be used in connection with the sale of securities, (6) all accountants' fees, attorneys' fees and other expenses connected with any of the foregoing, and (7) all other noninsurance expenses of the same kind or character. Notwithstanding the generality of the foregoing, ArMA shall not be obliged to pay any expenses involved in the conduct of an insurance business by MICA, nor any of MICA's routine and continuing corporate expenses, including but not limited to (a) expenses relating to the filing of annual statements with the Arizona Corporation Commission, the Department of Insurance and other regulatory bodies, (b) the expenses of members and trustees' meetings, and (c) legal, accounting and actuarial fees relating to such corporate matters.

2. **Possibility of Unintended Interpretation.** As thusly worded Paragraph 3.A. may be interpreted as providing that ArMA shall pay certain of such expenses indefinitely, which interpretation was not intended.

3. **Necessity of Clarifying Former Agreement.** In order to eliminate such possible misinterpretation and clarify the original intent that ArMA shall assume and pay only the initial organizational expenses of MICA, it is necessary that such paragraph be revised.

Agreement:

4. **Modification of Former Agreement.** In order to accomplish the purposes set forth in Paragraph 3 hereof, the parties agree that for all purposes whatsoever Paragraph 3.A. of that certain FINANCING AND SERVICE AGREEMENT of February 24, 1976, as modified as of March 18, 1976, shall be construed to read in its entirety as follows:

"**A. Payment of Organization Expenses.** ArMA shall forthwith and hereafter pay and discharge, as they accrue, (1) all expenses which may have heretofore accrued in connection with the organization and incorporation of MICA, (2) all expenses of MICA connected with its application for and the issuance to it of a Certificate of Authority authorizing it to carry on and conduct a professional liability (malpractice) insurance business in the State of Arizona, (3) all expenses of MICA in connection with the filing by it of an application for the registration of securities and the issuance to it of a permit authorizing the sale of securities in the State of Arizona pursuant to the Securities Act of Arizona, (4) all expenses of MICA in connection with any application for and issuance of a license as a dealer in its own securities, (5) all printing expenses in connection with the printing of the initial prospectus or offering circular necessary to be used in connection with the sale of securities, (6) all accountants' fees, attorneys' fees and other expenses connected with any of the foregoing, and (7) all other initial noninsurance expenses of the same kind or character. Notwithstanding the generality of the foregoing, ArMA shall not be obliged to pay any fees or expenses connected with the continuation of (as distinguished from the initial obtaining of) any aforementioned authorization, permit or license; any expenses involved in the conduct of an insurance business by MICA; nor any of MICA's routine and continuing corporate expenses, including but not limited to (a) expenses relating to the filing of annual statements with the Arizona Corporation Commission, the Department of Insurance and other regulatory bodies, (b) the expenses of members and trustees' meetings, and (c) legal, accounting and actuarial fees relating to such corporate maters."

5. **Republication of Agreement.** All other terms and conditions of the FINANCING AND SERVICE AGREEMENT of February 24, 1976, as modified as of March 18, 1976, none of which shall be construed to have been in any manner modified by any implication hereof, shall remain in full force and effect.

IN WITNESS WHEREOF, the parties hereto have caused this instrument to be executed as of its date by the duly authorized officers.

Mr. Robinson pointed out that essentially this modified agreement puts and end time on when ArMA is to pay for such things as reprinting prospectus etc., which was not clearly set forth in the original agreement.

SENATE BILL 1132

The Chairman reviewed the letter from the Arizona Board of Osteopathic Examiners in Medicine and Surgery regarding their concern for provisions of the subject bill. RECEIVED.

ARIZONA OPHTHALMOLOGISTS SOCIETY

The recently started educational campaign by the subject society regarding optometrists was reviewed. RECEIVED.

OPINION POLL

Mr. Robinson reviewed the status of the membership and non-membership opinion poll. He indicated that the cut off date was 8 27 76 and final count figures would be completed soon thereafter for presentation to the Board of Directors at their meeting October 2, 1976.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS, THAT SHOULD A SUBSTANTIAL MAJORITY VOTE AGAINST MAINTAINING UNIFIED MEMBERSHIP, THAT A SPECIAL

MEETING OF THE HOUSE OF DELEGATES BE CALLED AS SOON AS POSSIBLE, PERHAPS NOVEMBER 20, 1976, FOR THE PURPOSE OF VOTING ON A BYLAWS CHANGE DOING AWAY WITH UNIFIED MEMBERSHIP WITH THE AMA.

COMMITTEE ON CONTINUING PHYSICIAN EDUCATION

Dr. Sattenspiel discussed the concept of changing the Scientific Assembly Committee to the subject committee with the expansion of their responsibilities to include the provision of continuing education programs in addition to the annual convention. These courses would be primarily for those physicians outside the Phoenix and Tucson area.

This matter to be reviewed again in the future.

ARIZONA DEPARTMENT OF HEALTH SERVICES

Swine Flu Program

Dr. Dandoy reviewed the forthcoming immunization program planned for this fall. She indicated that the various counties will be handling the program differently, that is, some will be having special days while others will be conducting clinics over a longer period of time. Regarding Federal Liability Legislation she offered the following statement:

"All physicians should be aware that there are several options available to each physician who gives monovalent or bivalent vaccine containing A New Jersey 76 (HswIN1):

1. The physician who volunteers his time as an attending physician at a public community clinic is protected.
2. The physician who gives vaccine in his own office or clinic, does not charge for the vaccine or its administration and uses the federally approved informed consent form provided by the Arizona Program is protected by the federal legislation.
3. The physician who gives vaccine in his own office or clinic and charges for its administration and or does not use the federally approved informed consent form is not protected by the federal liability act.

The full text of the legislation is contained in the Congressional Record - House, August 10, 1976."

Heavy Metals Study

Dr. Dandoy reported that the subject study has been concluded and that there will soon be a release on the findings of the study.

Auto Emission Inspection

Dr. Dandoy pointed out that repeal of the subject program will be on the November ballot.

IT WAS MOVED AND CARRIED TO TAKE THIS MATTER TO THE BOARD OF DIRECTORS WITH THE RECOMMENDATION THAT THE ASSOCIATION TAKE A PUBLIC STANCE AGAINST THE REPEAL EFFORT.

CORRESPONDENCE

Arizona Board of Regents

The letter dated 8 18 76 from the Board of Regents which stated that they could not accept our offer to help with the U of A College of Medicine Department of Surgery matter was received for information.

George W. King, M. D., letter 8/14/76

Dr. King's letter regarding our supporting the U. S. Savings Bond Program was received for information.

OTHER BUSINESS

Medical Opinion

Mr. Robinson reported that KOOL-TV had cancelled the subject TV show due to scheduling changes. RECEIVED.

Next Meeting

It was determined to have the next Executive Committee meeting on Friday, October 1st.

Characteristics of Physicians

It was determined not to participate in the funding of the AMA's program to publish a series of brochures on the *Characteristics of Physicians* for each state.

AMA Board of Trustees

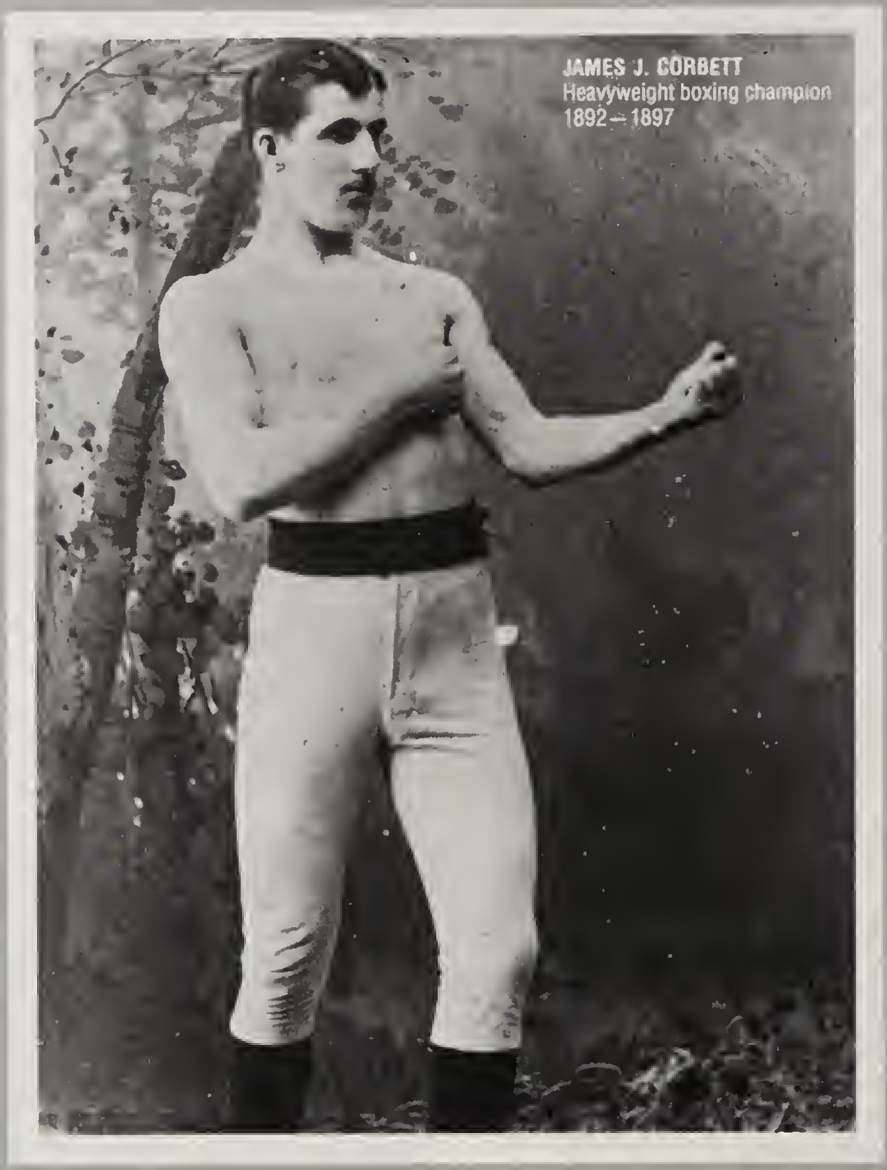
IT WAS MOVED AND CARRIED TO APPROVE A ONE NIGHT HOSPITALITY SUITE FOR DR. CLOUD AT THE AMA CLINICAL CONVENTION AS WELL AS CONTINUING TO SUPPORT HIS REELECTION AT THE JUNE, 1977 MEETING.

Meeting adjourned 8:37 p. m.

William E. Crisp, M. D.
Secretary

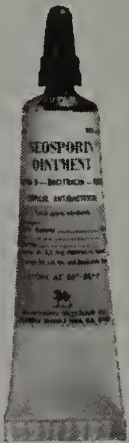
In spite of the improvements that have been made, once a doctor obtains a license in a state, it is his for life. Unless he is guilty of violating laws, he does not have to show that he has continued to keep up with advances in medicine, he does not have to prove his continuing competence to maintain his license . . . Bulletin, American College of Surgeons, Sept. 1974, Vol. 59, #9.

Famous Fighters



NEOSPORIN® Ointment
(polymyxin B-bacitracin-neomycin)
is a famous fighter, too.

Provides overlapping, broad-spectrum antibacterial action to help combat infection caused by common susceptible pathogens (including staph and strep).



Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 units; zinc bacitracin 400 units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

INDICATIONS: Therapeutically (as an adjunct to systemic therapy when indicated) for topical infections, primary or secondary, due to susceptible organisms, as in: • infected burns, skin grafts, surgical incisions, otitis externa • primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia) • secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis) • traumatic lesions, inflamed or suppurating as a result of bacterial infection.

Prophylactically, the ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing. **CONTRAINDICATIONS:** Not for use in the eyes or external ear canal if the eardrum is perforated. This product is contraindicated in those individuals who have shown hypersensitivity to any of the components. **WARNING:** Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended. **PRECAUTIONS:** As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs. **ADVERSE REACTIONS:** Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

I Cannot Tell A Lie—It Does Taste Like BANANAS!"

When acute, non-specific diarrhea causes the stomach to revolt, the tasteful counterattack is Donnagel®-PG. Donnagel-PG provides all the benefits of paregoric and—instead of that unpleasant paregoric taste—a delicious banana flavor good enough to make even an expert flip his wig.

Now with child-proof closure

Donnagel®-PG Ⓥ

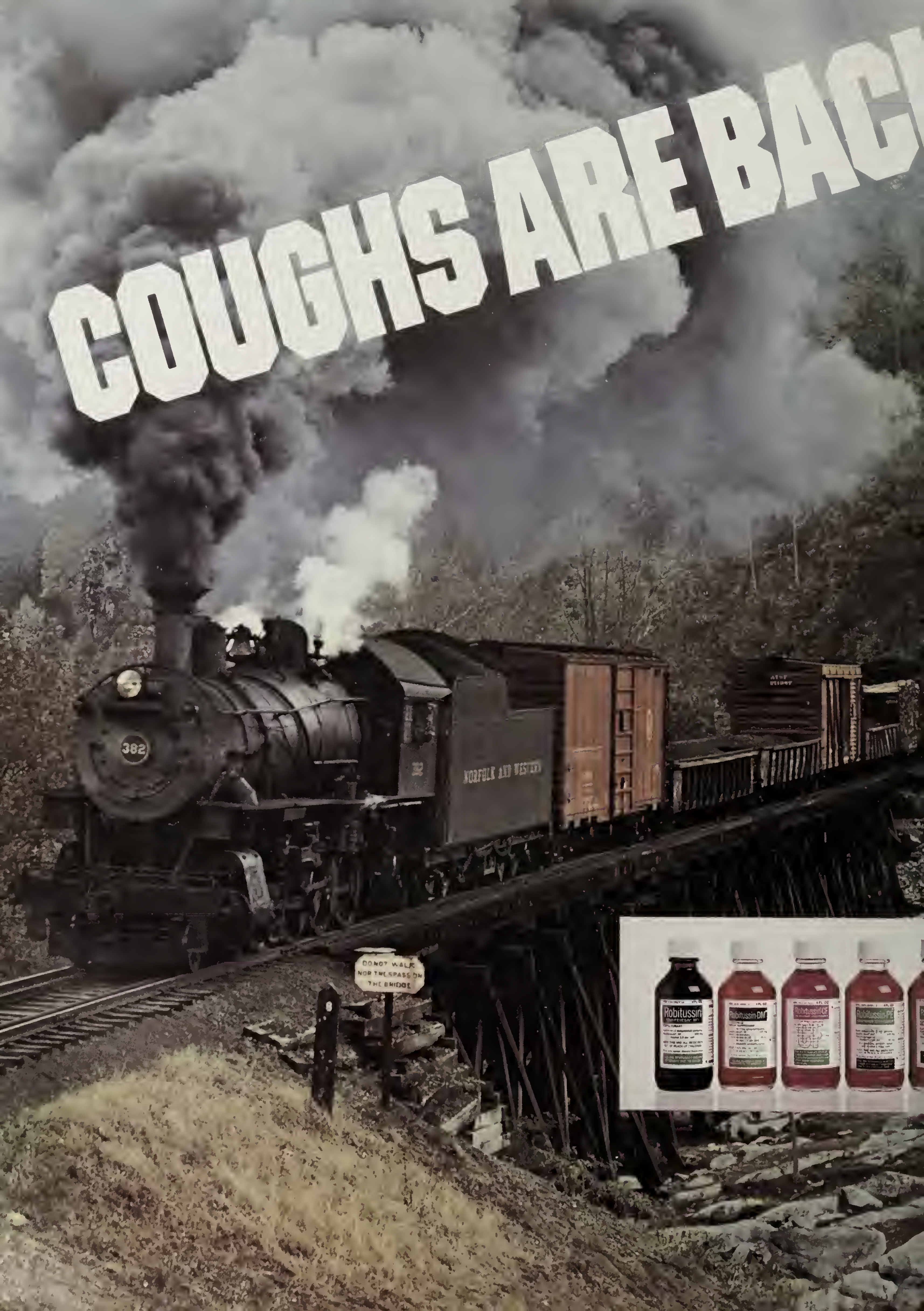
Donnagel with paregoric equivalent

For diarrhea

Each 30 ml. contains:

Kaolin	6.0 g.
Pectin	142.8 mg.
Hyoscyamine sulfate	0.1037 mg.
Atropine sulfate	0.0194 mg.
Hyoscine hydrobromide	0.0065 mg.
Powdered opium, USP (equivalent to paregoric 6 ml.) (warning: may be habit forming)	24.0 mg.
Sodium benzoate (preservative)	60.0 mg
Alcohol, 5%	

A·H·ROBINS A.H Robins Company, Richmond, Virginia 23220 Member of Certified Medical Representatives Institute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NORFOLK AND WESTERN
DO NOT WALK NOR TRESPASS ON THE BRIDGE
Robitussin
Robitussin-DM
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Robitussin-PE

CLEAR THE TRACT

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lu" and u.r.i.—
lear the tract
ith the famous
obitussin® Line!

e 5 members of the
bitussin® family all contain
e expectorant, guaifenesin,
help clear the lower
spiratory tract. Guaifenesin
orks systemically to help
imulate the output of lower
spiratory tract fluid. This
ıhanced flow of less viscid
ecretions promotes ciliary
ction and makes thick,
spissated mucus less viscid
ıd easier to raise. As a
sult, dry, unproductive
oughs become more
oductive and less frequent.

*R PHOTO: Norfolk & Western Branch Train
202 west bound near Alvarado, Va (Oct., 1956).
s line reaches the highest point of any railroad
t of the Rockies (elevation 3,577 ft.) with a
imum grade of 3%. It crosses 108 bridges,
ne 700 ft. long! Photo by O. Winston Link.*

For productive and unproductive coughs

Robitussin®

Each 5 ml teaspoonful contains:

Guaifenesin, NF	100 mg
Alcohol, 3.5%	

For severe coughs

Robitussin A-C® Ⓥ

Each 5 ml teaspoonful contains:

Guaifenesin, NF	100 mg
Codeine Phosphate, USP	10.0 mg
(warning: may be habit forming)	
Alcohol, 3.5%	

Non narcotic for 6-8-hour cough control

Robitussin-DM®

Each 5 ml teaspoonful contains:

Guaifenesin, NF	100 mg
Dextromethorphan Hydrobromide, NF	15 mg
Alcohol, 1.4%	

Decongests nasal passages and sinus openings as it helps relieve coughs

Robitussin-PE®

Each 5 ml teaspoonful contains:

Guaifenesin, NF	100 mg
Pseudoephedrine Hydrochloride, NF	30 mg
Alcohol, 1.4%	

Decongestant action helps control cough and clear stuffy noses and sinuses. Non narcotic.

Robitussin-CF®

Each 5 ml teaspoonful contains:

Guaifenesin, NF	50 mg
Phenylpropanolamine Hydrochloride, NF	12.5 mg
Dextromethorphan Hydrobromide, NF	10 mg
Alcohol, 1.4%	

All Robitussin formulations available on your Rx or Recommendation.

For many years Robins has spotlighted the expectorant action of the Robitussin cough formulations by featuring action photographs of steam engines like the one on the preceding page. In keeping with this tradition, last year the company commissioned a well-known illustrator to render full-color drawings of several classic locomotives . . . accurate to the minutest detail. Chances are you requested and received the first locomotive in this series, The William Mason, last winter. Now, the second one is available. (See below). To order your print suitable for framing, write "Robitussin Clear-Tract Engine #2" on your Rx pad and mail to "Vintage Locomotives," Dept. T4,
A. H. Robins Company,
1407 Cummings Drive,
Richmond, Va. 23220.

The Davis Camel (1873)

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CONTRIBUTIONS

The Editor sincerely solicits contributions of scientific articles for publication ARIZONA MEDICINE. All such contributions are greatly appreciated. All will be given equal consideration.

Material submitted for publication in ARIZONA MEDICINE should conform to the following policies:

1. Manuscripts, including references or bibliography, should be typewritten, double-spaced, on one side of the paper only, and the original and a carbon enclosed.

2. Be guided by the general rules of medical writing as followed by the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.

3. Although the Editors try to catch inaccuracies, the ultimate responsibility is the author's.

4. Articles are accepted for publication only if they are contributed exclusively to this Journal. Ordinarily, contributors will be notified within 60 days if a manuscript is accepted for publication. Every effort will be made to return unused manuscripts.

5. The Journal reserves the right to edit all material.

6. Reprints will be supplied to the author at printing cost.

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

THE FOLLOWING INSTITUTIONS HAVE RECEIVED ArMA ACCREDITATION FOR CONTINUING MEDICAL EDUCATION.

ARIZONA STATE HOSPITAL, PHOENIX
DESERT SAMARITAN HOSPITAL, MESA
GOOD SAMARITAN HOSPITAL, PHOENIX
PHOENIX INDIAN MEDICAL CENTER
MARICOPA COUNTY GENERAL HOSPITAL, PHOENIX
MEMORIAL HOSPITAL, PHOENIX
ST. LUKE'S HOSPITAL AND MEDICAL CENTER, PHOENIX
ST. JOSEPH'S HOSPITAL AND MEDICAL CENTER, PHOENIX
TUCSON HOSPITALS MEDICAL EDUCATION PROGRAM, TUCSON
VETERANS ADMINISTRATION CENTER, PRESCOTT

CONTINUING MEDICAL EDUCATION ACTIVITIES SPONSORED BY THESE INSTITUTIONS RECEIVE CATEGORY 1 CREDIT FOR THE ArMA CERTIFICATE IN CONTINUING MEDICAL EDUCATION AND THE AMA PHYSICIAN'S RECOGNITION AWARD.

DISTRICT VIII MEETING OF THE AMERICAN COLLEGE OF OBSTETRICIANS & GYNECOLOGISTS

October 10-14, 1976
The Adams Hotel, Phoenix, AZ

SPONSOR: District VIII, American College of Obstetricians & Gynecologists

CONTACT:
David Pent, M.D.
2021 N. Central
Phoenix, AZ 85004

Approved for 27 required hours toward the ArMA Certificate in Continuing Medical Education.

DERMATOLOGY CLINICAL CONFERENCE

October 18, 1976 — 5:15 p.m.
Tucson Medical Center, Tucson, AZ

SPONSOR: U of A College of Medicine
Dept. of Internal Medicine,
Dermatology Section

CONTACT:
Peter Lynch, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

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PEDIATRIC LUNG DISEASE SEMINAR

October 21, 1976
Little America, Flagstaff, AZ

SPONSOR: Arizona Lung Association

CONTACT:
Ben Chaiken
1239 E. McDowell
Phoenix, AZ 85006

Approved for 6 required hours toward the ArMA Certificate in Continuing Medical Education.

THIRD ANNUAL MEETING OF THE SOCIETY FOR THE ADVANCEMENT OF FREESTANDING AMBULATORY SURGICAL CARE

October 29-31, 1976
Continental Plaza Hotel, Chicago, Ill.

SPONSOR: Society for the Advancement of
Freestanding Ambulatory Surgical Care

CONTACT:
Boyden L. Crouch, M.D.
1040 E. McDowell Road
Phoenix, AZ 85013

Approved for 6 required hours toward the ArMA Certificate in Continuing Medical Education.

24TH ANNUAL MEETING OF THE MEDICAL SOCIETY OF THE UNITED STATES AND MEXICO

November 10-13, 1976
Puerto Vallarta, Mexico
Holiday Inn

SPONSOR: Medical Society of the United
States and Mexico

CONTACT:
Mrs. Virginia E. Bryant
Executive Secretary
333 W. Thomas Rd., Ste 207
Phoenix, AZ 58013

BASIC SCIENCE REVIEW SEMINARS

November 16, 1976
December 21, 1976
7-9:30 p.m., Arizona Medical Center, Rm. 3505

SPONSOR: U of A College of Medicine

CONTACT:
George D. Comerci, M.D.
Office of Continuing Medical
Education & Outreach
U of A College of Medicine
Tucson, AZ 85724

THIRD INTERNATIONAL COCCIDIOIDOMYCOSIS SYMPOSIUM

November 17-19, 1976
Tucson, AZ

SPONSOR: American College of Chest
Physicians, U of A, Arizona
Regional Medical Program

CONTACT:
American College of Chest Physicians
911 Busse Highway
Park Ridge, Illinois 60068

AMERICAN ASSOCIATION FOR CLINICAL IMMUNOLOGY AND ALLERGY

November 28-December 2, 1976
Braniff Place Hotel, Tucson, AZ

SPONSOR: American Association for
Clinical Immunology & Allergy

CONTACT:
Howard Silber, Exec. Dir.
American Assoc. for Clinical
Immunology & Allergy
104 S. 39th St.
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Omaha, Nebraska 68101

GASTROINTESTINAL DIAGNOSIS

December 4 & 5, 1976
Maricopa County General Hospital
Phoenix, AZ

SPONSOR: Department of Radiology

CONTACT:
Department of Radiology
Maricopa County General Hospital
2601 East Roosevelt
Phoenix, AZ 85008

Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

STAFF QUARTERLY MEETING

December 15, 1976
Chimney's Restaurant
Glendale, AZ

SPONSOR: Glendale Samaritan Hospital

CONTACT:
Gerald Marshall, M.D.
6010 W. Northern
Glendale, AZ 85301

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CLINICAL IMMUNOLOGY, ALLERGY AND RHEUMATOLOGY ROUNDS

Every Friday Noon-1 p.m.

Every Friday Noon-1 p.m.

SPONSOR: U of A College of Medicine
Dept. of Internal Medicine,
Clinical Immunology Section

CONTACT:
John Boyer, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

ENDOCTRINOLOGY SEMINAR

Every Thursday, Noon-1 p.m.
1st, 3rd, & 5th Thursday — Rm. N318, VA Hospital
2nd & 4th Thursday, Rm. 6505,
Tucson Medical Center

SPONSOR: U of A College of Medicine
Department of Internal Medicine

CONTACT:
Charles Nugent, M.D.
U of A College of Medicine
Tucson, AZ 85721

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HEMATOLOGY-ONCOLOGY CLINICAL CONFERENCE

Every Tuesday, Noon-1 p.m.
1st, 3rd, & 5th Tuesdays — Rm. 6505,
AZ Medical Center
2nd & 4th Tuesdays — Rm. N318,
Veterans Adm. Hospital

SPONSOR: U of A College of Medicine, Dept. of
Internal Medicine

CONTACT:
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U of A College of Medicine
Tucson, AZ 85721

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GRAND WARD ROUNDS — TRAUMA

Every Tuesday, 8 a.m.
Arizona Medical Center, Tucson, AZ

SPONSOR: U of A College of Medicine,
Surgery Dept., Trauma Section

CONTACT:
Martin Silverstein, M.D.
U of A College of Medicine
Tucson, AZ 85721

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MEDICAL GRAND ROUNDS

Every Wednesday, Noon-1 p.m.
1st, 3rd, & 5th Wednesday — Staff Conf. Rm., VA Hospital
2nd & 4th Wednesday — Rm 5403, Arizona Medical Center

SPONSOR: U of A College of Medicine
Dept. of Internal Medicine

CONTACT:
Jay Smith, M.D.
U of A College of Medicine
Tucson, AZ 85721

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PROBLEM CASE WORKSHOPS

3rd Monday of each month 7:30 a.m.
Room 4410, Arizona Medical Center, Tucson, AZ

SPONSOR: Division of Ophthalmology, U of A
College of Medicine

CONTACT:
H. E. Cross, M.D., Ph.D.
Arizona Medical Center
Dept. of Surgery
Tucson, AZ

Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

PSYCHIATRIC GRAND ROUNDS

Every Wed., Sept. to May, 4-5:30 p.m.
Rm. 8403, Arizona Medical Center, Tucson, AZ

SPONSOR: U of A College of Medicine
Dept. of Psychiatry

CONTACT:
Alan Levenson, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 11/2 required hour per session toward the ArMA Certificate in Continuing Medical Education.

TRAUMA CONFERENCE

Every Monday, 4 p.m.
Rm. 4410, Arizona Medical Center, Tucson, AZ

SPONSOR: U of A College of Medicine
Dept. of Surgery, Trauma Section

CONTACT:
Martin Silverstein, M.D.
U of A College of Medicine
Tucson, AZ 85721

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STAFF EDUCATION CONFERENCE

Wednesdays, Weekly, 1 p.m.
Arizona State Hospital, Phoenix, AZ

SPONSOR: Arizona State Hospital

CONTACT:
Howard E. Wulsin, M.D.
Arizona State Hospital
2500 E. Van Buren
Phoenix, AZ 85008

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SURGICAL GRAND ROUNDS 4TH TUESDAY OF EACH MONTH

Hospital Auditorium
Baptist Hospital, Phoenix

SPONSOR: Baptist Hospital Phoenix

CONTACT:
James B. Shields, M.D.
6036 N. 19th Ave.
Phoenix, AZ 85015

Approved for 1½ required hours per month toward the ArMA Certificate in Continuing Medical Education.

PATIENT STAFFING CONFERENCE

Three times weekly
Camelback Hospital, Phoenix, AZ

SPONSOR: Camelback Hospital

CONTACT:
Stuart M. Gould, Jr., M.D.
Medical Director
Camelback Hospital
5055 N. 34th St.
Phoenix, AZ 85018

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CAMELBACK HOSPITAL CLINICAL CONFERENCE

3rd Tuesday monthly
Camelback Hospital, Phoenix, AZ

SPONSOR: Camelback Hospital

CONTACT:
Stuart M. Gould, Jr., M.D.
Medical Director
Camelback Hospital
5055 N. 34th St.
Phoenix, AZ 85018

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COUNTER TRANSFERENCE GROUP

Weekly, Thurs. 8-10 p.m.

SPONSOR: Phoenix Psychiatric Council

CONTACT:
James E. Campbell, M.D.
5051 N. 34th St.
Phoenix, AZ 85018

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DESERT SAMARITAN HOSPITAL
Wednesday Evenings 7 p.m.

November 16 — Management of Carcinoma of the Breast. Tue. Evening—Fiesta Inn
December 1 Neonatal Meningitis

SPONSOR: Desert Samaritan Hospital

CONTACT:
L. A. Rosati, M.D.
Laboratory
Desert Samaritan Hospital
Mesa, AZ 85202

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GLENDALE SAMARITAN HOSPITAL MEDICAL EDUCATION LECTURES
January through December, 1976, 7 a.m.

SPONSOR: Glendale Samaritan Hospital

CONTACT:
Gerald Marshall, M.D.
6010 W. Northern
Glendale, AZ 85301

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PULMONARY DISEASE GRAND ROUNDS
Mondays — 12 Noon
D-5 North Conference Rm., Good Samaritan Hospital, Phoenix, AZ

SPONSOR: Pulmonary Disease Teaching Service
Good Samaritan Hospital

CONTACT:
Bernard E. Levine, M.D.
Pulmonary Function Laboratory
Good Samaritan Hospital
1033 E. McDowell Hospital
Phoenix, AZ 85006

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CLINICAL CANCER CONFERENCE
3rd Wednesday Every Month
Butler Bldg. Conference Room
Good Samaritan Hospital, Phoenix, AZ

SPONSOR: Good Samaritan Hospital

CONTACT:
John A. Bruner, M.D.
926 E. McDowell Road
Phoenix, AZ 85006

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BI-MONTHLY MEDICAL EDUCATION SEMINAR
Every other Wed. AM
Begin 7/3/74
Maryvale Samaritan Hospital, Phoenix, AZ

SPONSOR: Medical Staff Maryvale Hospital

CONTACT:
Thomas J. Groves, M.D.
6037 W. Elm St.
Phoenix, AZ 85033

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TUMOR BOARD CONFERENCE
Every Friday at Noon
Kiva Conference Room
Phoenix Memorial Hospital

SPONSOR: Phoenix Memorial Hospital

CONTACT:
Howard Kimball, M.D.
333 West Thomas Road
Phoenix, AZ 85013

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MONTHLY MEDICAL EDUCATION SEMINAR
Third Monday of the Month
Kiva Conference Room
Phoenix Memorial Hospital

SPONSOR: Medical Staff of Memorial Hospital

CONTACT:
George Scharf, M.D.
1201 South 7th Avenue
Phoenix, AZ 85007

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Last Tues. of Month
Plaza International, Tucson, AZ

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FAMILY PRACTICE CONFERENCE
1st Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Dir. of Medical Education
7300 E. 4th St.
Scottsdale, AZ

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MORBIDITY & MORTALITY CONFERENCE
2nd Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

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Scottsdale, AZ

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CLINICAL PATHOLOGICAL CONFERENCE

4th Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

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MEDICAL GRAND ROUNDS

3rd Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

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Dir. of Medical Education
7300 E. 4th St.
Scottsdale, AZ

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CARDIOLOGY CONFERENCE

Weekly — Friday 8-9 a.m.
St. Mary's Hospital Auditorium
Tucson, AZ

SPONSOR: St. Mary's Hospital

CONTACT:
A. L. Forte, M.D.
St. Mary's Hospital
Tucson, AZ 85724

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GRAND ROUNDS

Each Thursday 7 a.m.-8 a.m.
St. Mary's Hospital and Health Center,

SPONSOR: Depts. of Medicine, Surgery, Radiology, Pathology and Family Practice.

CONTACT:
Richard Silver, M.D.
Chairman, Medical Education
and Library Committee
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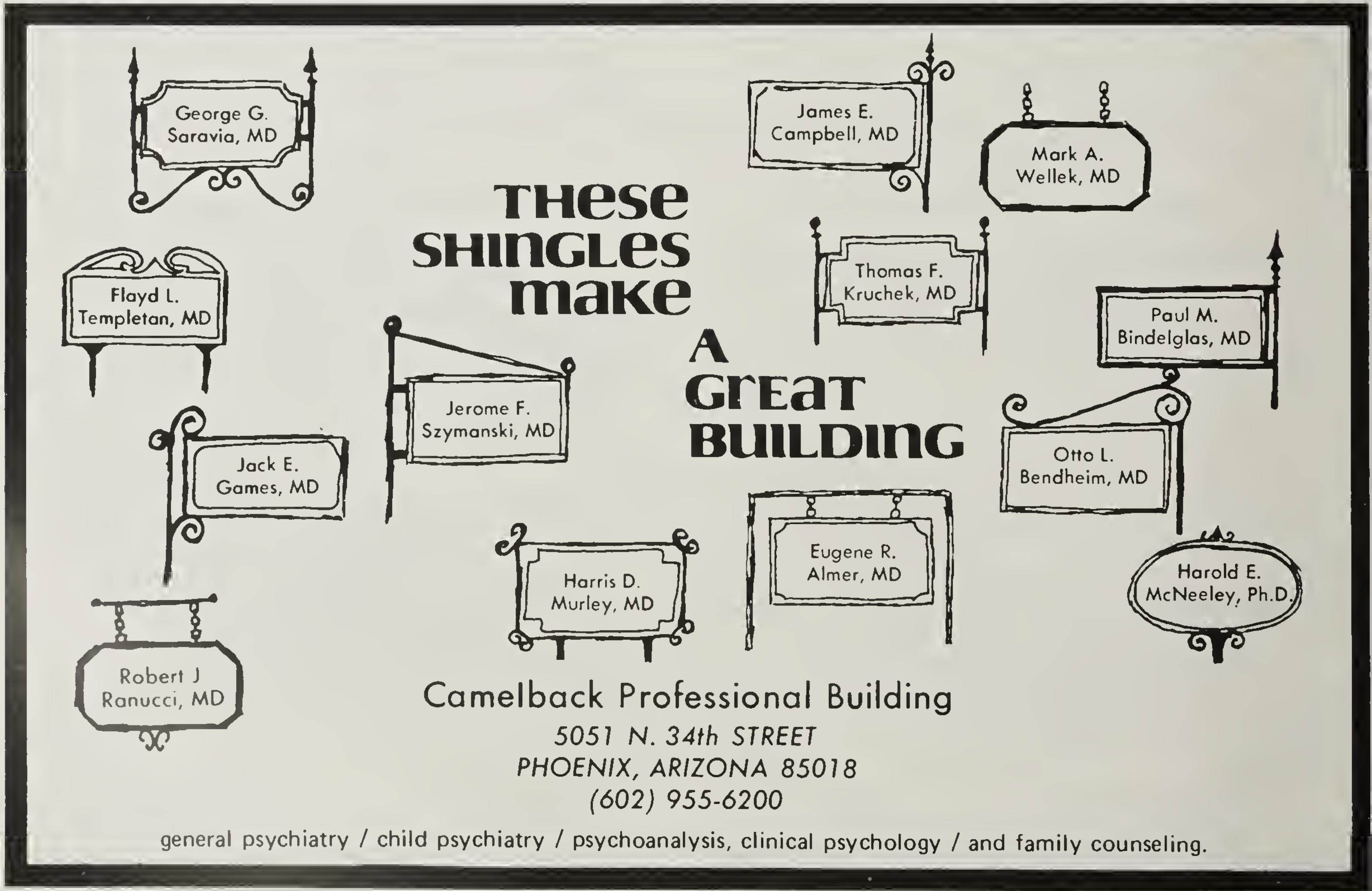

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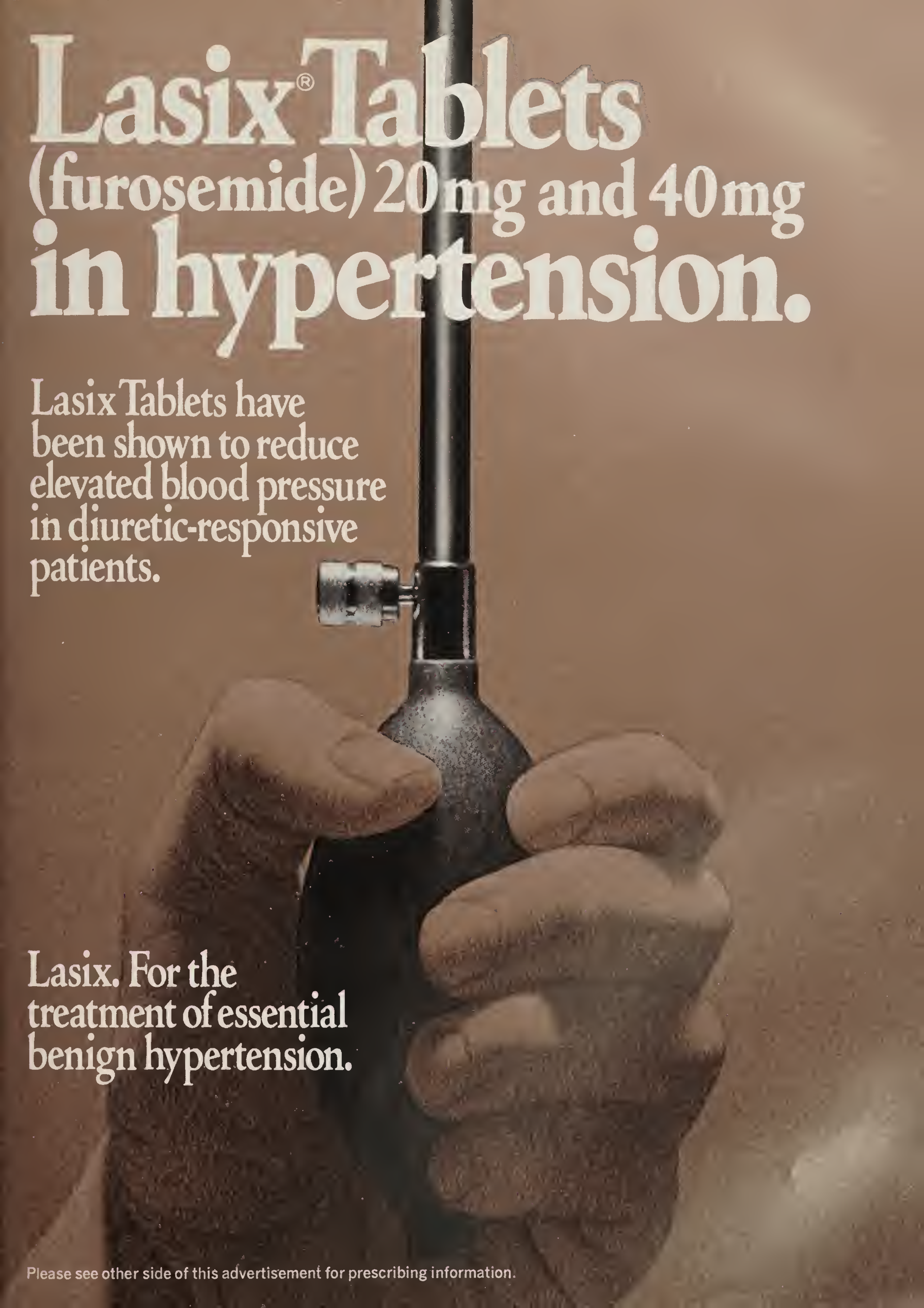

Lasix® Tablets
(furosemide) 20 mg and 40 mg
in hypertension.

Lasix Tablets have been shown to reduce elevated blood pressure in diuretic-responsive patients.

Lasix. For the treatment of essential benign hypertension.

Please see other side of this advertisement for prescribing information.

Lasix® Tablets (furosemide) 20 mg and 40 mg in hypertension.

A brief summary of the Prescribing Information for

Lasix® (furosemide) Tablets 20 mg and 40 mg

WARNING—Lasix (furosemide) is a potent diuretic which if given in excessive amounts can lead to a profound diuresis with water and electrolyte depletion. Therefore, careful medical supervision is required, and dose and dose schedule have to be adjusted to the individual patient's needs. (See under "Dosage and Administration.")

Indications—Lasix (furosemide) is indicated for the treatment of the edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Hypertension—Lasix (furosemide) may be used for the treatment of hypertension alone or in combination with other antihypertensive drugs. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controllable with Lasix (furosemide) alone.

CONTRAINDICATIONS—Because animal reproductive studies have shown that Lasix (furosemide) may cause fetal abnormalities, the drug is contraindicated in women of childbearing potential. (See "Additional Information.")

Lasix (furosemide) is contraindicated in anuria. If increasing azotemia and oliguria occur during treatment of severe progressive renal disease, the drug should be discontinued. In hepatic coma and in states of electrolyte depletion, therapy should not be instituted until the basic condition is improved or corrected. Lasix (furosemide) is contraindicated in patients with a history of hypersensitivity to this compound.

Warnings—Excessive diuresis may result in dehydration and reduction in blood volume, with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients. Excessive loss of potassium in patients receiving digitalis glycosides may precipitate digitalis toxicity. Care should also be exercised in patients receiving potassium depleting steroids.

Frequent serum electrolyte, CO_2 and BUN determinations should be performed during the first few months of therapy and periodically thereafter, and abnormalities corrected or the drug temporarily withdrawn.

In patients with hepatic cirrhosis and ascites, initiation of therapy with Lasix (furosemide) is best carried out in the hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma; therefore, strict observation is necessary during the period of diuresis. Supplemental potassium chloride and, if required, an aldosterone antagonist are helpful in preventing hypokalemia and metabolic alkalosis.

Patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

In those instances where potassium supplementation is required, an oral liquid preparation should be used rather than enteric-coated potassium salts.

There have been several reports, published and unpublished, concerning nonspecific small-bowel lesions consisting of stenosis, with or without ulceration, associated with the administration of enteric-coated thiazides with potassium salts. These lesions may occur with enteric-coated potassium tablets alone or when they are used with nonenteric-coated thiazides, or certain other oral diuretics.

These small-bowel lesions have caused obstruction, hemorrhage, and perforation. Surgery was frequently required, and deaths have occurred.

Available information tends to implicate enteric-coated potassium salts, although lesions of this type also occur spontaneously. Therefore, coated potassium-containing formulations should be administered only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Patients with known sulfonamide sensitivity may show allergic reactions to Lasix (furosemide).

Precautions—As with any potent diuretic, electrolyte depletion may occur during therapy with Lasix (furosemide), especially in patients receiving higher doses and a restricted salt intake. Electrolyte depletion may manifest itself by weakness, dizziness, lethargy, leg cramps, anorexia, vomiting, and/or mental confusion.

Asymptomatic hyperuricemia can occur and gout may rarely be precipitated. Reversible elevations of BUN may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

When parenteral use of Lasix (furosemide) precedes its oral use, it should be kept in mind that cases of tinnitus and reversible hearing impairment have been reported. There have also been some reports of cases in which irreversible hearing impairment occurred. Usually, ototoxicity has been reported when Lasix (furosemide) was injected rapidly in patients with severe impairment of renal function at doses exceeding several times the usual recommended dose and in whom other drugs known to be ototoxic were often given. If the physician elects to use high dose parenteral therapy in patients with severely impaired renal function, controlled intravenous infusion is advisable (for adults, it has been reported that an infusion rate not exceeding 4 mg Lasix [furosemide] per minute has been used).

Increases in blood glucose, and alterations in glucose tolerance tests with abnormalities of the fasting and two-hour postprandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Lasix (furosemide) may lower serum calcium levels, and rare cases of tetany have been reported.

Patients receiving high doses of salicylates, in conjunction with Lasix (furosemide) may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Diuretics such as furosemide may enhance the nephrotoxicity of cephaloridine. Therefore, Lasix (furosemide) and cephaloridine should not be administered simultaneously.

Sulfonamide diuretics have been reported to decrease arterial responsiveness to pressor amines and to enhance the effect of tubocurarine. Great caution should be exercised in administering curare or its derivatives to patients undergoing therapy with Lasix (furosemide), and it is advisable to discontinue Lasix (furosemide) for one week prior to any elective surgery.

Adverse Reactions—Various forms of dermatitis, including urticaria and rare forms of exfoliative dermatitis, erythema multiforme, pruritus, paresthesia, blurring of vision, postural hypotension, nausea, vomiting, or diarrhea.

Anemia, leukopenia, aplastic anemia, and thrombocytopenia (with purpura). Rare cases of agranulocytosis which responded to treatment.

In addition, the following rare adverse reactions have been reported; however, relationship to the drug has not been established with certainty: sweet taste, oral and gastric burning, paradoxical swelling, headache, jaundice, thrombophlebitis and emboli and acute pancreatitis.

Lasix (furosemide)-induced diuresis may be accompanied by weakness, fatigue, lightheadedness or dizziness, muscle cramps, thirst, increased perspiration, urinary bladder spasm, and symptoms of urinary frequency.

Dosage and Administration

ADULTS

The usual adult dose of Lasix (furosemide) is 20 to 80 mg given as a single dose.

If the diuretic response with a single dose of 20 to 80 mg is not satisfactory, the following schedule should be used: Increase this dose by increments of 20 or 40 mg not sooner than 6 to 8 hours after the previous dose until the desired diuretic effect has been obtained. This individually determined single dose should then be given once or twice daily. The dose of Lasix (furosemide) may be carefully titrated up to 600 mg per day in those patients with severe clinical edematous states.

With doses exceeding 80 mg/day and given for prolonged periods, careful clinical and laboratory observations are particularly advisable.

Hypertension—The usual dose of Lasix (furosemide) is 40 mg twice daily both for initiation of therapy and for maintenance. Careful observations for changes in blood pressure must be made when this compound is used with other antihypertensive drugs, especially during initial therapy. The dosage of other agents must be reduced by at least 50 percent as soon as Lasix (furosemide) is added to the regimen to prevent excessive drop in blood pressure. As the blood pressure falls under the potentiating effect of Lasix (furosemide), a further reduction in dosage, or even discontinuation, of other antihypertensive drugs may be necessary. It is further recommended, if 40 mg twice daily does not lead to a clinically satisfactory response, to add other hypotensive agents, e.g., reserpine, rather than to increase the dose of Lasix (furosemide).

INFANTS AND CHILDREN

Pediatric Administration: The usual initial dose of oral Lasix in infants and children is 2 mg/kg body weight, given as a single dose. If the diuretic response is not satisfactory after the initial dose, dosage may be increased by 1 or 2 mg/kg not sooner than 6 to 8 hours after the previous dose. Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

How Supplied—Lasix Tablets 40 mg (furosemide) supplied as white, round, monogrammed, scored tablets.

Lasix Tablets 20 mg (furosemide) supplied as white, oval, monogrammed tablets.

Note: Dispense in dark containers. Exposure to light may cause slight discoloration which, however, does not alter potency.

Additional Information

Toxicology

The acute toxicity of Lasix (furosemide) has been determined in mice, rats, and dogs. In all three animal species, the oral LD_{50} of Lasix (furosemide) exceeded 1000 mg/kg of body weight, while the intravenous LD_{50} ranged from 300 to 680 mg/kg. Intragastric injection of the drug in newborn rats resulted in an LD_{50} of 380 mg/kg.

The acute toxicity of high doses of Lasix (furosemide) was characterized by convulsions, paralysis, and collapse. Surviving animals often became dehydrated and depleted of electrolytes due to the diuresis induced by Lasix (furosemide). In the newborn rats, intragastric injection of the drug caused hyperactivity and anorexia.

Chronic toxicity studies with Lasix (furosemide) were done in rats and dogs. In a one-year study in rats, renal tubular degeneration occurred, with all doses higher than 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day). A six-month study in dogs revealed calcification and scarring of the renal parenchyma at all doses above 10 mg/kg (83 percent of the maximal recommended human dose of 600 mg per day).

Reproductive Studies

The effects of Lasix (furosemide) on embryonic and fetal development and on pregnant dams were studied in mice, rats, and rabbits.

Lasix (furosemide) caused unexplained maternal deaths and abortions in the rabbit when 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day) was administered between days 12 to 17 of gestation. In a previous study the lowest dose of only 25 mg/kg (2 times the maximal recommended human dose of 600 mg per day) caused maternal deaths and abortions. In a third study, none of the pregnant rabbits survived a dose of 100 mg/kg. Data from the above studies indicate fetal lethality which can precede maternal deaths.

The results of the mouse study and one of the three rabbit studies also showed an increased incidence of hydronephrosis (distention of the renal pelvis and, in some cases, of the ureters) in fetuses derived from treated dams as compared to the incidence in fetuses from the control group.

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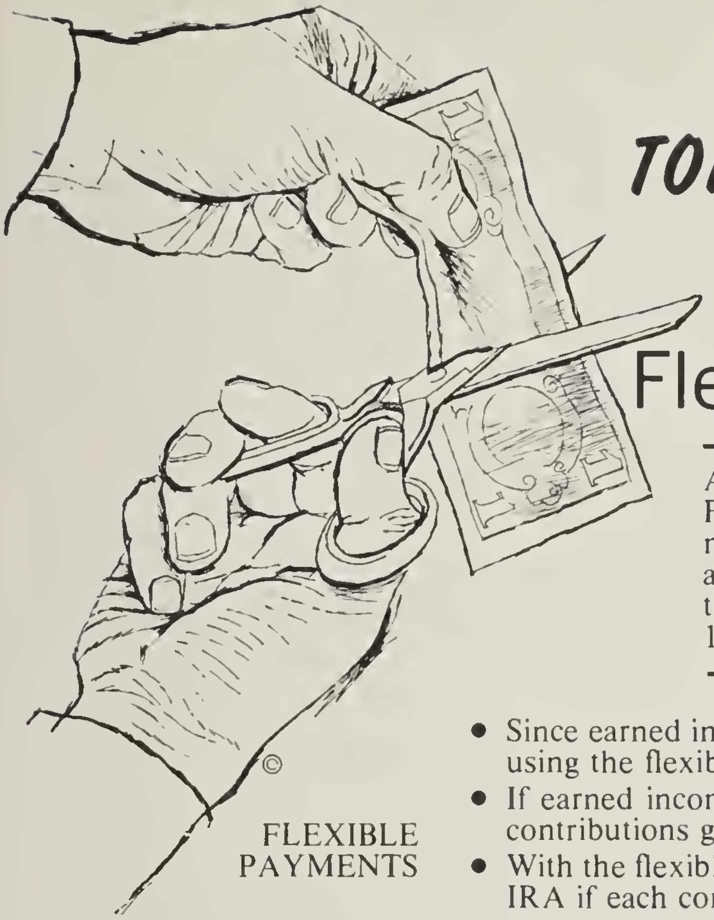

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- Since earned income may fluctuate, you can adjust annual contributions by using the flexible payment feature.
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Your dollars will accumulate on a guaranteed growth basis. In addition, we are currently paying a non-guaranteed, higher interest rate than the guaranteed amount. The following illustrates both the guaranteed and current non-guaranteed accumulation and income at age 65.

Assume $100 monthly contributions to age 65
Monthly Benefit at 65 for 120 months certain and life

Age at Issue	Guaranteed Dollar Accumulation	Current (Non-Guar.) Accumulation	Monthly Benefit Guaranteed Male	Guaranteed Female	Non-Guaranteed Male	Non-Guaranteed Female
35	$58,458	$102,476	$364	$330	$818	$764
45	32,164	45,264	205	186	361	337
55	13,523	15,492	86	78	123	115

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If you would like more information, an HBA Life agent will be glad to discuss our FPRA with you. Please phone us at the number below.

THE HBA LIFE INSURANCE COMPANY
1337 N. FIRST ST. (P.O. BOX 1272), PHOENIX, AZ 85001 (602) 258-4885

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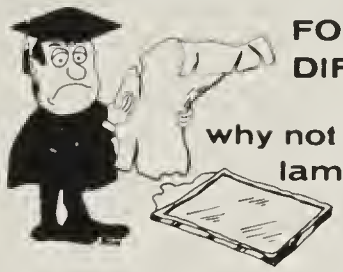

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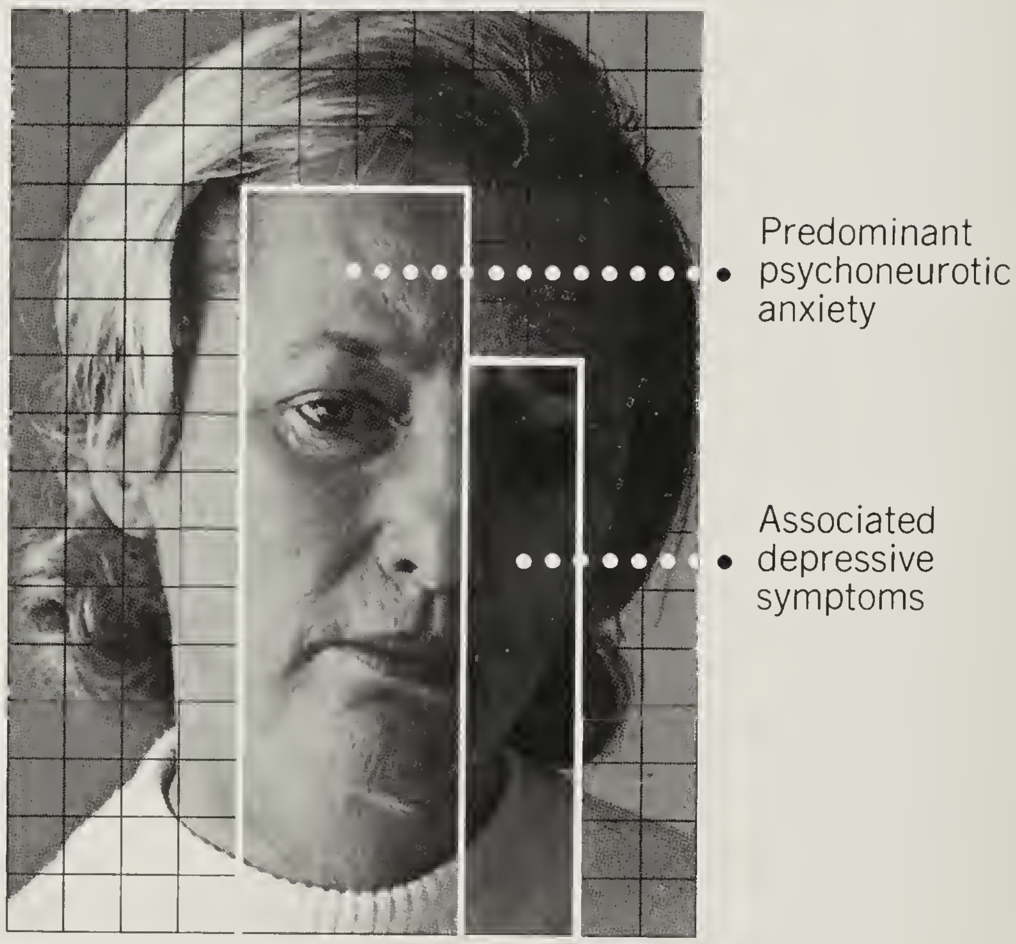
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Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequen and/or severity of grand mal seizures m require increased dosage of standard an convulsant medication; abrupt withdraw may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ir gestion of alcohol and other CNS depres sants. Withdrawal symptoms (similar to those with barbiturates and alcohol) hav occurred following abrupt discontinuanc (convulsions, tremor, abdominal and mu cle cramps, vomiting and sweating). Kee addiction-prone individuals under carefu

respond to one

According to her major
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xiety. But according to the
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is is because her problem,
hough primarily one of ex-
sive anxiety, is often accom-
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logy. Valium (diazepam)
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excessive anxiety is re-
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ns associated with it are also
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There are other advan-
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ic anxiety with secondary
pressive symptoms: the
chotherapeutic effect of
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ovement is usually apparent
in the patient within a few days rather than in a week or two, although it may take longer in some patients. In addition, Valium (diazepam) is generally well tolerated; as with most CNS-acting agents, caution patients against hazardous occupations requiring complete mental alertness.

Also, because the psychoneurotic patient's symptoms are often intensified at bedtime, Valium can offer an additional benefit. An *h.s.* dose added to the *b.i.d.* or *t.i.d.* treatment regimen can relieve the excessive anxiety and associated depressive symptoms and thus encourage a more restful night's sleep.

Valium®
(diazepam) Ⓘⓥ
2-mg, 5-mg, 10-mg scored tablets

in psychoneurotic
anxiety states
with associated
depressive symptoms

veillance because of their predisposi-
to habituation and dependence. In
gnancy, lactation or women of child-
ring age, weigh potential benefit
inst possible hazard.

cautions: If combined with other psy-
tropics or anticonvulsants, consider
efully pharmacology of agents em-
ved; drugs such as phenothiazines,
cotics, barbiturates, MAO inhibitors
other antidepressants may potentiate
ction. Usual precautions indicated in
ents severely depressed, or with latent
ression, or with suicidal tendencies.
Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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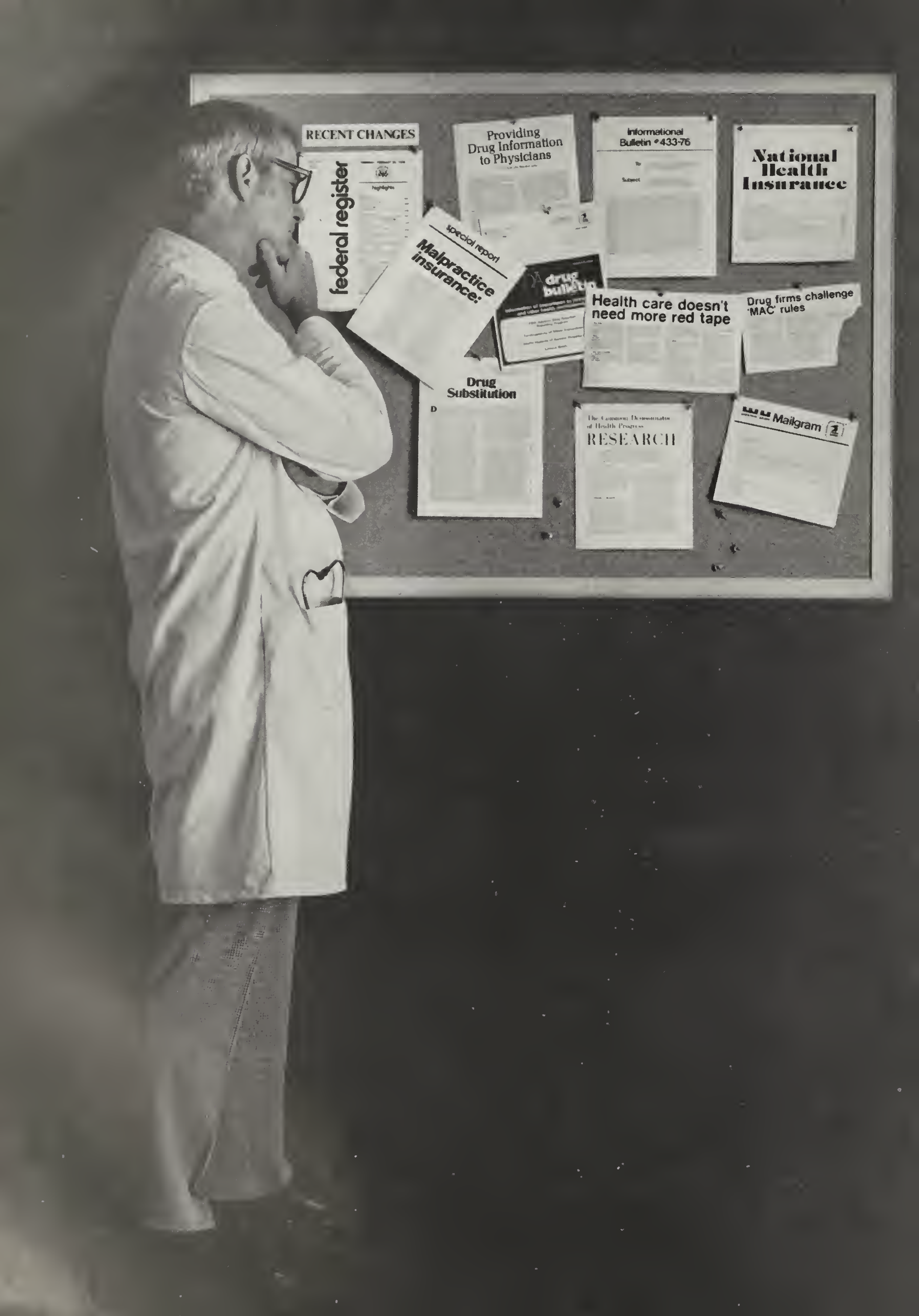
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THERE ARE A LOT OF PEOPLE GETTING BETWEEN YOU AND YOUR PATIENT.

Medicine today is in the spotlight, subjected to all kinds of scrutiny. Your control over patient therapy is being monitored, judged and occasionally abrogated, sometimes by unknown third parties.

The worry is that in the wake of this focus, the relationship between you and your patient will be weakened, without offsetting benefits. Consider three examples:

Drug substitution In most states, pharmacy laws, regulations or professional custom stipulate that your non-generic prescriptions be filled with the precise products you prescribe. But in the last five years, a dozen or more State laws have been changed, permitting the pharmacist in most cases to select a product of the same generic drug to fill any prescription.

Ironically, this dilution of physician control has taken place against a background of growing evidence that purportedly equivalent drug products may be inequivalent, since neither present drug standards nor their enforcement are optimal. In fact, the FDA itself says it has not enforced the same standards for hundreds of "follow-on" products that it had applied to the original NDA approvals. Thus physician control over patient therapy is being eroded with a risk that patients may be exposed to drugs of uncertain quality.

The major advertised claim for substitution is reduced prescription prices for consumers. Yet no documentation of any significant savings has been produced.

MAC Maximum Allowable Cost, MAC for short, is a Federal regulation designed to cut the Government's drug bill by setting price ceilings for drugs dispensed to Medicare and Medicaid patients. Unless the prescriber certifies on the prescription that a particular product is medically necessary, the Government intends to pay only for the cost of the lowest-priced, purportedly-equivalent, generally-available product. The effect of the program may be that elderly and indigent patients will be restricted to products which someone in Washington believes are priced right. Practicing doctors will have little to say about administration of the program, since Government will have absolute authority to make its choices stick.

The drug lag The future of drug and device research depends upon a scientific and regulatory environment that encourages therapeutic innovations. The American pharmaceutical industry annually is spending more than $1 billion of its own funds and evaluating more than 1,200 investigational compounds in clinical research. Disease targets include cancer, atherosclerosis, viruses and central nervous system disorders, among others. But there is a major barrier to the flow of new drugs to your patients: The cost of the research is more than ten times what it was, per product, in 1962; and whereas governmental clearance of new drug applications took six months then, it commonly consumes two years now.

The FDA needs adequate time, of course, to consider data. But it is equally clear that the present approval process contributes to needless delay of needed therapy. That's why the increased efficiency of the drug approval process is vital to all our futures.

If these issues concern you, we suggest that you make your voice heard—among your colleagues and your representatives in State legislatures and in Washington.

It could make a difference in your practice tomorrow.

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2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.
3. Threatened abortion.

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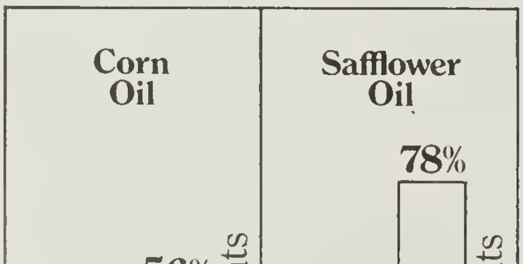

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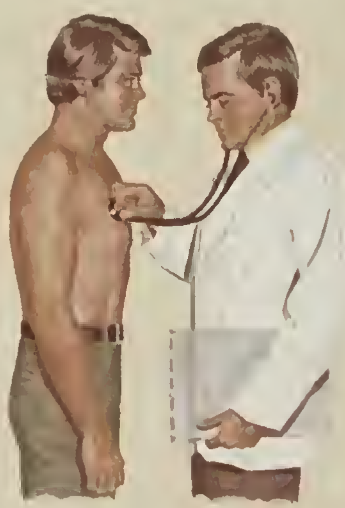
Narrowing down pneumonias

Upjohn

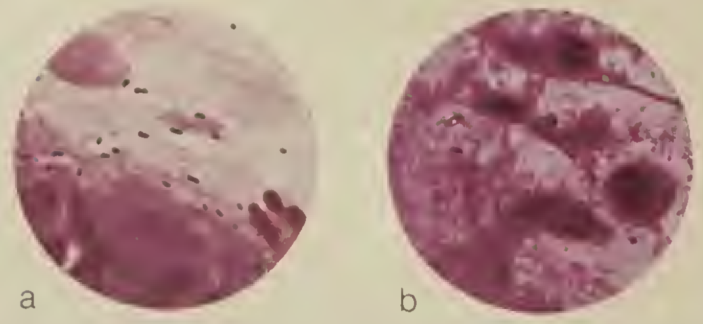
Bacterial or nonbacterial?

In the early stages of mild to moderate pneumonia, it is often difficult to reach an accurate bacteriologic diagnosis. A tentative differentiation may be made on history-taking, physical examination, CBC with differential analysis, and microscopic study of the sputum smear. Specimens should be obtained for culture and sensitivity testing, but when a bacterial pneumonia is diagnosed, therapy is often instituted before the etiological agent is positively identified.



Bacterial pneumonias may have a sudden onset with a shaking chill, rapid development of fever, pleuritic pain, and cough productive of rust-colored sputum. The Gram-stained sputum smear generally shows polymorphonuclear neutrophils as well as a predominance of the causative organisms. These are likely to be *Streptococcus pneumoniae,* still by far the most frequently encountered agent in bacterial pneumonia.[1] The CBC reveals marked leukocytosis with a shift to the left.

In *nonbacterial pneumonias*—mycoplasmal or viral—classical symptoms tend to develop more slowly, with myalgia, lassitude, and headache predominating. Sputum production is usually scanty, and the sputum smear is relatively uninformative, showing gram-positive cocci and other organisms which are part of the normal pharyngeal flora. The leukocyte count is normal or slightly elevated.



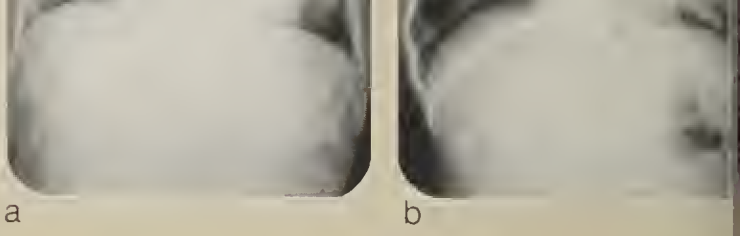
Direct Gram-stained sputum smears (a) Pneumococcal pneumonia—note abundant polymorphonuclear leukocytes, as well as gram-positive diplococci. (b) Nonspecific—consistent with viral or mycoplasmal pneumonia Note large mononuclear cell, as well as a few polymorphs and mixed bacterial flora (pharyngeal contaminants).

Mycoplasmal or viral?

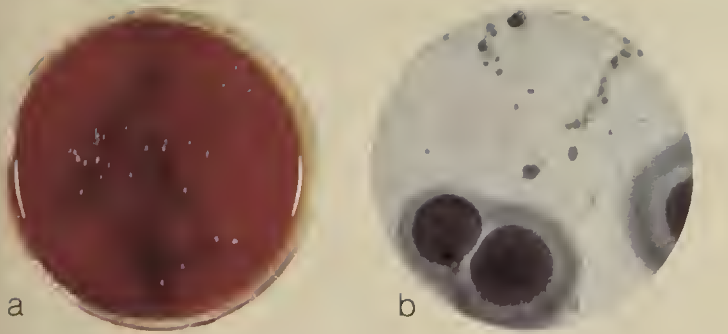
Differentiation between mycoplasmal and viral pneumonias may be impossible in the acute stage

Serologic testing and culture methods for *Mycoplasma pneumoniae* are complex and time-consuming, taking as long as two weeks after obtaining samples. The sensitivity and specificity c the test for cold agglutinins have been questioned. The complement-fixing antibody test may reflect previous infection. Furthermore, facilities for culturi *M. pneumoniae* are not widely available.[3]

If treatment is to be initiated, therefore, it may b necessary to start on the basis of a *presumptive* diagnosis of mycoplasmal pneumonia.[1-3] In reachi such a diagnosis, the physician relies on clinical judgment, considering such factors as the age of th patient and the history of exposure. For example, *Mycoplasma pneumoniae* is considered the most common cause of pneumonia among ambulatory patients aged 20 to 35.[1]



Chest x-rays of patients with (a) pneumococcal pneumonia—classically heavy, extensive infiltration of left lung; (b) mycoplasmal pneumonia—mild infiltrate confined to left lower lobe. Roentgenography usually does not help in differential diagnosis, since both types of pneumonias may present with a wide spectrum of x-ray as well as clinical findings.



(a) Distinct mucoid colonies of type 3 pneumococci *(Streptococcus pneumoniae)* on sheep blood agar showing greenish discoloration (alpha-hemolysis) of medium. (b) Typical "fried-egg" colonies of *Mycoplasma pneumoniae* consisting of dense central core with lighter periphery. Cultural and serologic methods for detecting *M. pneumoniae* are complex, time-consuming, and not widely available.

Expectant therapy

n the patient with a presumptive diagnosis of nycoplasmal pneumonia or bacterial pneumonia, it nay be desirable to initiate antibiotic therapy before ulture and sensitivity results are available.

A course of erythromycin or tetracycline is onsidered effective in the treatment of mycoplasmal neumonia to help speed the clearing of infiltrate and horten the duration of symptoms.[1,3] In pneumococcal neumonia, erythromycin is an effective alternative to enicillin, the drug of choice. A recent report, based n data from 200 hospitals of 100 beds or more, found 8% of *S. pneumoniae* sensitive *in vitro* to rythromycin.[4]

Among these therapeutic agents, only rythromycin provides effective coverage of both *Aycoplasma pneumoniae* and *S. pneumoniae*. The enicillins are not effective against *Mycoplasma*, and *. pneumoniae* has shown a relatively high incidence f resistance to tetracycline.

When erythromycin is selected for therapy, -Mycin (erythromycin enteric-coated tablets, Upjohn) s a good choice. E-Mycin is administered and bsorbed as active erythromycin base, and may be aken q.i.d., q 6h, or b.i.d., immediately after meals or etween meals. Thus, patients can use mealtimes to elp them remember their medication. The enteric oating on E-Mycin tablets helps ensure efficient bsorption in the intestinal tract, and bioavailability tudies show that E-Mycin can be expected to roduce predictable, acceptable blood levels. The ow cost of E-Mycin helps assure economical therapy.

E-Mycin rarely causes serious side effects and is ot associated with liver toxicity.* The most frequent ide effects are upper gastrointestinal, such as bdominal cramping and discomfort, and are dose-elated. Nausea, vomiting, and diarrhea occur nfrequently with usual oral doses. Serious allergic eactions, including anaphylaxis, have rarely been eported.

---

*Use cautiously in patients with severe liver impairment

REFERENCES: 1. Chusid EL, Dalrymple W, Holloway WJ, et al: Managing the infectious neumonias. *Patient Care* 9:122-167, 1975. 2. The occasional might of mycoplasma. *mergency Med* 7:82, 85, 1975. 3. Stevens DA: Viral and Mycoplasma pneumonias. *ostgrad Med* 55:81-86, 1974. 4. Data source: PMR Bacteriologic Report, Winter Series

Summary

Because pneumonias may be difficult to differentiate at the outset, treatment is often initiated before a causal diagnosis is made. However, readily available diagnostic criteria occasionally allow early differentiation between bacterial and nonbacterial pneumonias. When the diagnosis appears to be nonbacterial pneumonia, further differentiation between mycoplasmal pneumonia and viral pneumonia is more complex and time-consuming. Therefore, therapy is often initiated on the basis of a *presumptive* diagnosis of mycoplasmal pneumonia.

Erythromycin is an effective antibiotic against *Mycoplasma pneumoniae, Streptococcus pneumoniae*,† and *Streptococcus pyogenes*.† E-Mycin (erythromycin enteric-coated tablets, Upjohn) is administered and well absorbed as the active base, may be taken immediately after meals or between meals, and is essentially nontoxic.

†Although penicillin remains the drug of choice against these organisms erythromycin is an effective alternative

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erythromycin enteric-coated tablets, Upjohn

wide-ranging usefulness in pneumonia*

*Mild to moderately severe due to susceptible organisms

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1975-1976 Data are a compilation of laboratory reports submitted during December 1975 and January and February 1976 by 200 acute care hospitals of 100 beds or more

Please turn page for brief summary of prescribing information

Wide-ranging usefulness in pneumonia*

E-Mycin® 250 mg

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Upjohn

- For mild to moderately severe infections due to susceptible organisms that commonly invade the respiratory tract
- Essentially nontoxic (see Precautions and Adverse Reactions below)
- Documented bioavailability
- May be taken immediately after meals or between meals
- Active base formula produces predictable blood levels
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Alpha-hemolytic streptococci (viridans group): Short-term prophylaxis against bacterial endocarditis prior to dental or other operative procedures in patients with a history of rheumatic fever or congenital heart disease who are hypersensitive to penicillin. (Erythromycin is not suitable prior to genitourinary surgery where the organisms likely to lead to bacteremia are gram-negative bacilli or the enterococcus group of streptococci.)

Staphylococcus aureus: Acute infections of skin and soft tissue of mild to moderate severity. Resistance may develop during treatment.

Diplococcus pneumoniae: Upper respiratory tract infections (eg, otitis media, pharyngitis) and lower respiratory tract infections (eg, pneumonia) of mild to moderate degree.

Mycoplasma pneumoniae (Eaton agent, PPLO): In the treatment of primary atypical pneumonia, when due to this organism.

Treponema pallidum: Infections due to this organism.

Corynebacterium diphtheriae and *Corynebacterium minutissimum:* As an adjunct to antitoxin, to prevent establishment of carriers, and to eradicate the organism in carriers. In the treatment of erythrasma.

Entamoeba histolytica: In the treatment of intestinal amebiasi only. Extraenteric amebiasis requires treatment with other agents

Listeria monocytogenes: Infections due to this organism.

Contraindication: Contraindicated in patients with known hypersensitivity to erythromycin.

Warning: Safety for use in pregnancy has not been established

Precautions: Erythromycin is principally excreted by the liver Caution should be exercised in administering the antibiotic t patients with impaired hepatic function. Surgical procedure should be performed when indicated.

Adverse reactions: The most frequent side effects of erythromycin preparations are gastrointestinal, such as abdomina cramping and discomfort, and are dose-related. Nausea, vomiting, and diarrhea occur infrequently with usual oral doses During prolonged or repeated therapy, there is a possibility o overgrowth of nonsusceptible bacteria or fungi. If such infections occur, the drug should be discontinued and appropriate therapy instituted. Mild allergic reactions such as urticaria and other skin rashes have occurred. Serious allergic reactions, including anaphylaxis, have been reported.

Treatment of overdosage: The drug is virtually nontoxic, though some individuals may exhibit gastric intolerance to even therapeutic amounts. Allergic reactions associated with acute overdosage should be handled in the usual manner—that is, by the administration of adrenalin, corticosteroids, and antihistamines as indicated and the prompt elimination of unabsorbed drug in addition to all needed supportive measures.

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For additional product information, consult the package inser or see your Upjohn Representative.

NAS-4

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*Mild to moderately severe, due to susceptible organisms

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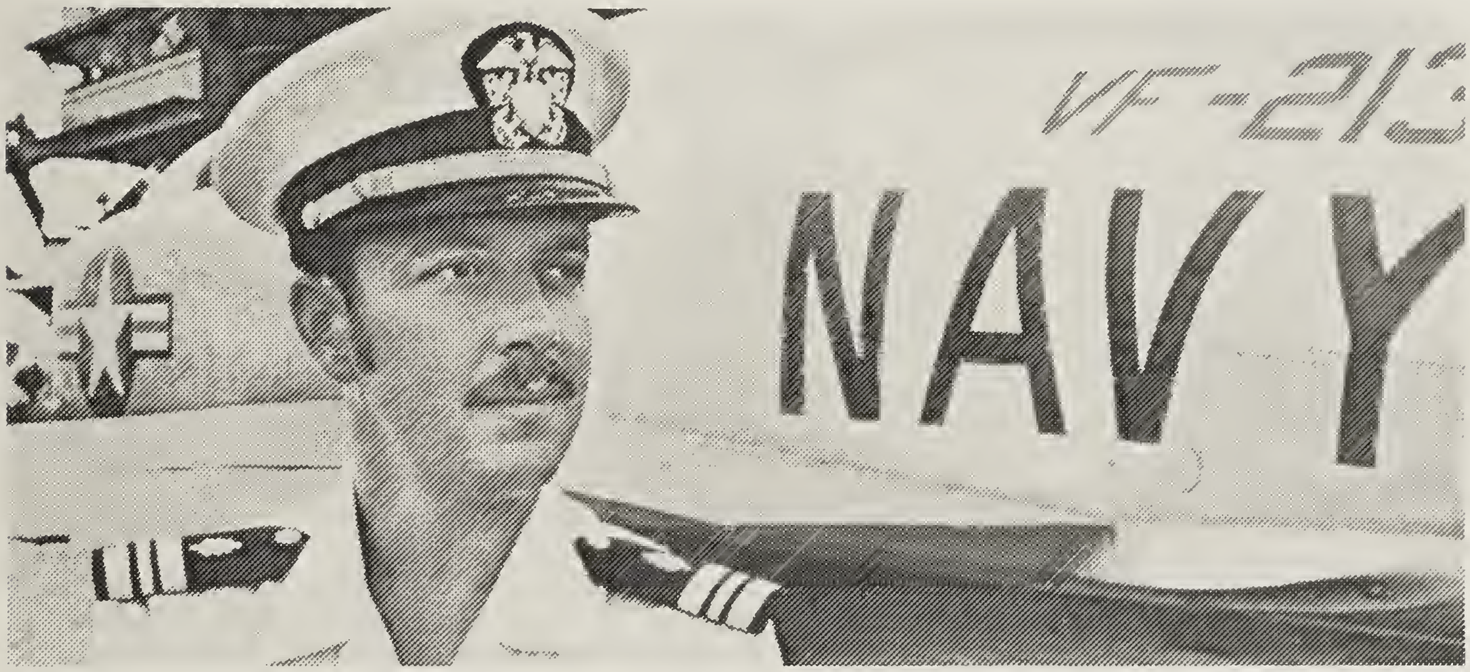

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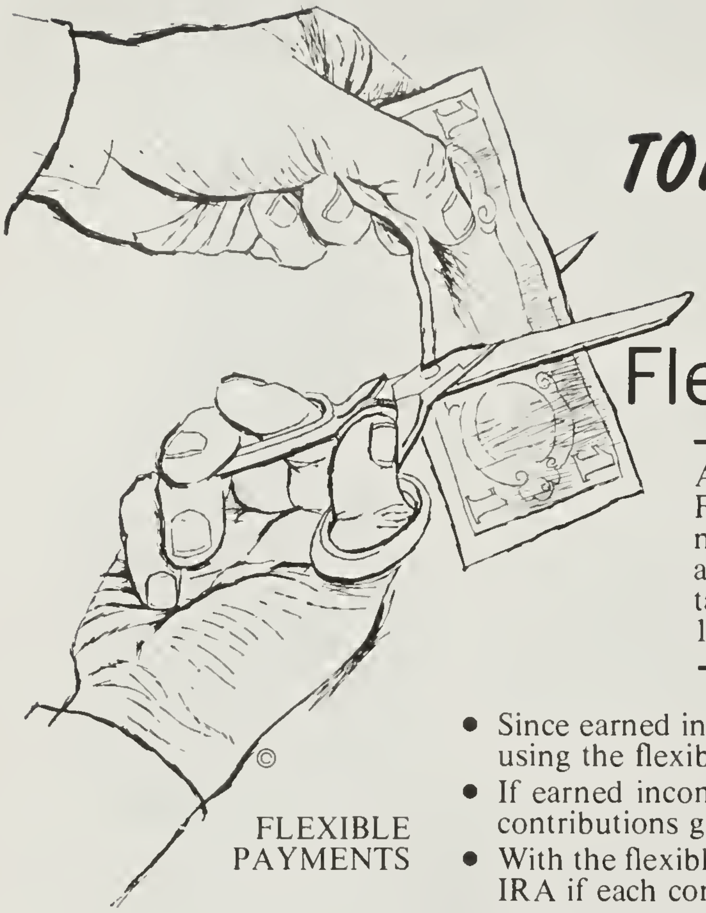

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Original Articles

OBJECTIVE PSYCHIATRIC AND PSYCHOLOGIC SIGNS OF BRAIN DISORDERS

HARRIS D. MURLEY, M.D.
DONALD R. MILAM, PH.D.
WARREN GORMAN, M.D., F.A.C.P.

There is no more mortifying experience to miss an organic (or systemic) brain malfunction, treating it as psychogenic. And the cost to the patient and his family can be inestimable.

This article describes simple screening devices with which any doctor can easily familiarize himself and which he can then employ.

SUMMARY

The relationship between minimal brain damage and learning disabilities in children, brain syndromes following strokes and certain treatable dementias in adults, is one of points on a continuum that is drawn by means of performing a minimal neurologic examination along with a minimal psychologic examination. This neurologic examination can be made by a non-neurologist, requires brief observations of the cranial nerves, motor and sensory status, an estimate of the intelligence from the vocabulary, plus ten simple observations for stance, coordination and verbal skills. The minimal "organic" psychologic examination which can be performed by a non-psychologist, observes impairment of the right cerebral hemisphere by the drawing of a Greek Cross and the Bender Visual-Motor Test, and impairment of the left cerebral hemisphere by the subtraction of Serial Sevens from 101 and the Wechsler Memory Scale. Techniques of testing are described.

The purpose of this article is to show that minimal brain dysfunction and learning disability in children, brain syndromes due to vascular occlusions, and certain treatable dementias such as Normal Pressure Hydrocephalus, are all points on a continuum which is delineated by simple neurological and psychological examinations that any medical practitioner can easily perform.

The scope of the problem covered by the Organic Brain Syndrome[1] is broad, as 35% of all patients 65 years of age and older who were discharged from general hospital psychiatric units showed a brain syndrome[16] and 30% of patients of any age on first admission to psychiatric hospitals showed a neurologic disorder.[8] The frequency of minimal brain dysfunction in all children is about 15%.[25]

In adults, the most common major clinical disorder causing the brain syndrome[9] is vascular occlusion, including hemorrhage and aneurysm, of the middle cerebral artery. Also to be noted is occlusion of the capsular branch of the posterior cerebral artery (which supplies the cranial nerve meclei that innervate the lips, tongue and pharynx), when its closure on the dominant side of the brain produces a characteristic dysarthria, in which the patient articulates words poorly, but can otherwise both emit and receive words satisfactorily. In multiple small lesions of both cerebral hemispheres, the control of the cranial nerve nuclei becomes deficient, producing "pseudobulbar palsy"[11] with a similar speech disorder, associated with[15,6] episodes of involuntary laughing and crying, mistakenly labeled hysterical.

> (Receptive aphasia, in contrast, is often associated with a lesion of the dominant superior temporal gyrus and adjacent parietal region. Unlike the receptive type, motor aphasia, in which the patient is able to receive speech symbols, but shows impairment in emitting them, is precipitated by a lesion on the dominant brain side, of the interior frontal gyrus. In mild or healing cases of aphasia, one often sees a diminution of verbal output both in speech and writing, with diminished reception or comprehension of verbal symbolic material. This adult condition is strikingly similar to the picture of learning disabilities in children.)

Other categories than vascular disease in adults merit differential diagnostic consideration:

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Reprint requests to Dr. Gorman, 5602 E. Nauni Valley Dr., Scottsdale, AZ 85243.

1. **Normal Pressure Hydrocephalus**

Normal pressure hydrocephalus appears in persons of either sex in their mature years before the senium, showing dementia,[7] gait disturbance and urinary incontinence.[3] Other features seen in this triad are behavior disorders, convulsive seizures, parkinsonism and akinetic mutism.[14] Neurological study shows hydrocephalus, but a normal cerebrospinal fluid pressure. Treatment consisting in running a shunt from the subordinate lateral ventricle to the peritoneum[10] produces a marked improvement in one-third of patients, therefore requiring consideration in all.

2. **Pseudodementia**

Pseudodementia describes a condition[21] that resembles the organic brain syndrome, but is not primarily due to disease of the brain. The patient, of either sex, often with depression, is middle aged or older, and before the illness may have a lower IQ than his peers. Neurologic and organic psychologic signs do not account for the symptoms. A mistaken diagnosis of "cerebral arteriosclerosis," when heard by the patient, increases his depression. Such patients do well with antidepressant treatment, especially electroconvulsive therapy, and on follow-up studies[20] show no more neurological signs than do control subjects of similar age and status.

NEUROLOGIC EXAMINATION

The medical literature contains frequent reviews on the "minimal neurological examination," for which the "practitioner's principal equipment is an ophthalmoscope and a prepared mind . . . Do not OMIT the mental status in which O is for orientation, M for memory, I for intelligence and T is for talk or speech."[5] The vocabulary usually provides a valid estimate of the intelligence.

In children with minimal brain dysfunction—which can be markedly improved by training and treatment[22,25]—a group of characteristic neurologic signs have been reported. While these signs are "minor" or "soft" signs compared to, say, dragging and circumducting the foot on walking, or differences in deep tendon reflexes, they are statistically significant[19] in patients with learning disabilities, of whom many respond to simple medication.[25] We have selected ten of these significant "minor" neurologic signs:

1. Stance. "Stand up, put your feet together and close your eyes. Put your arms straight out in front of you and spread your fingers, but don't touch your thumbs together." Abnormal if child 8 years or older does not understand, places arms elsewhere, or if arms drop or wander.

2. Fingers to thumb. Examiner holds up both hands, palms facing patient, who is asked to imitate examiner while the latter serially touches fingers to thumb, one hand at a time.

3. Hop.

4. Skip.

5. Alternating hand movements. Patient alternates placing palms on thighs, with backs of hands on thighs; first slowly, then rapidly.

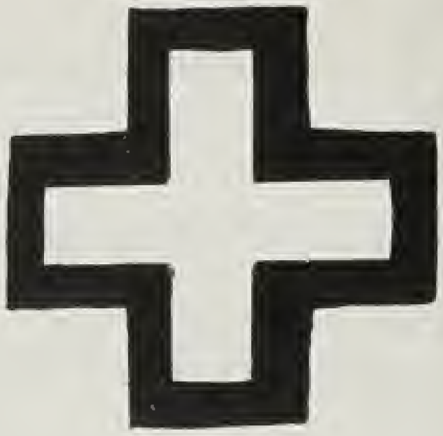
6. Right-left orientation. "Place your right hand on your left ear, left hand on your right knee . . ."

7. Wink each eye separately.

8. Say, "La, La" and "Da, Da" clearly and rapidly.

9. Spelling and writing.

10. Tonic neck reflex. With stance as in 1 above, use light finger pressure to rotate head 90 degrees, first to right, then to left, holding rotated position for a few seconds. Abnormal if arm on the occiput side flexes or lowers.[19]



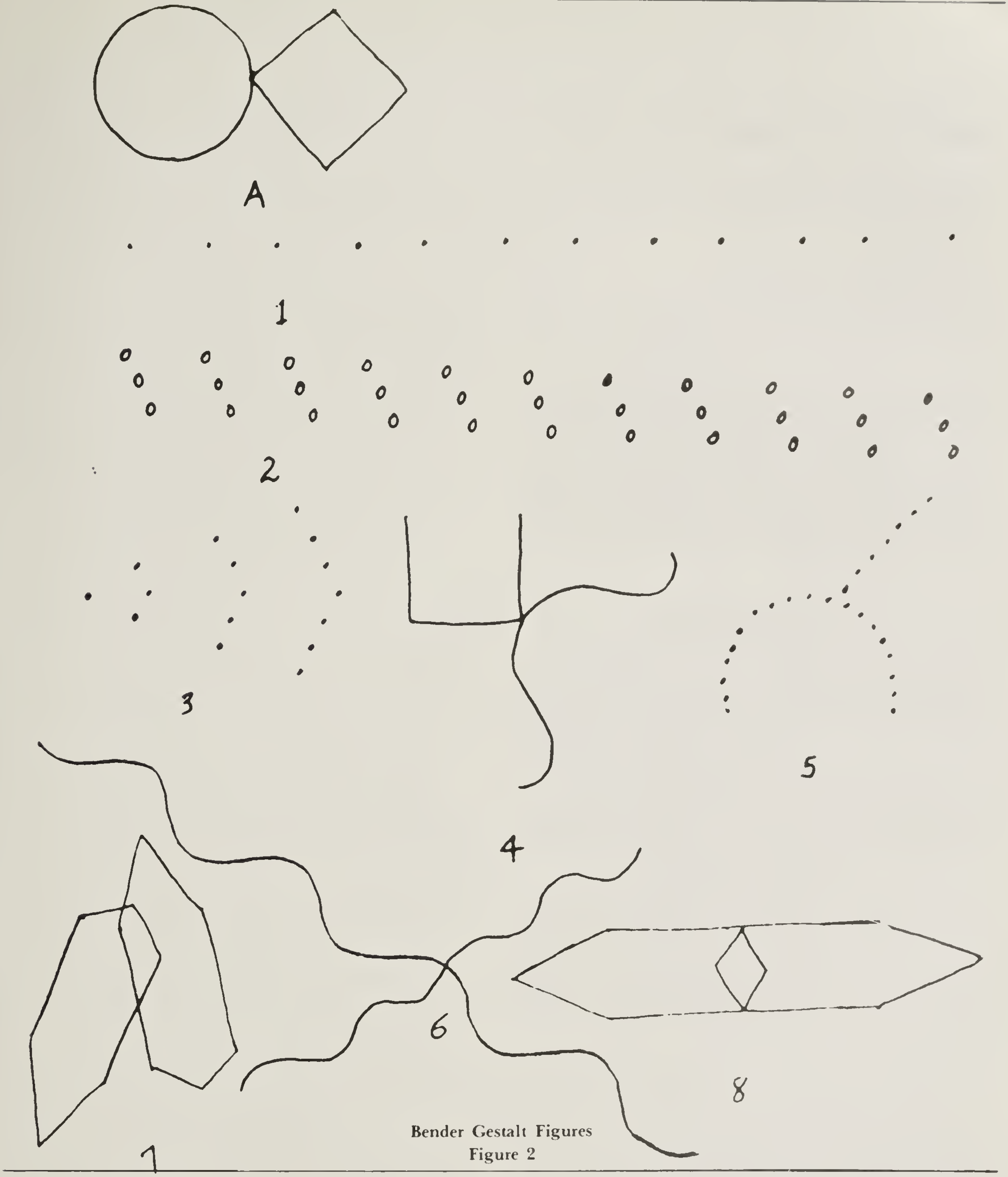
Greek Cross
Figure 1

MINIMAL PSYCHOLOGIC EXAMINATION

Psychological tests are useful both in screening, and confirming clinical tests for brain disorder,[18,23] because they reveal significant deficits in the language and memory function of the left cerebral hemisphere, and the auditory and visual form functions of the right cerebral hemisphere.[4]

Right Hemisphere

From among the many test instruments which lateralise impairment of the right hemisphere, we have chosen two drawing tests—the Greek Cross[12] and the Visual-Motor Test by Bender, often called the Bender Gestalt, as it depicts the



Bender Gestalt Figures
Figure 2

Gestalt or form, which represents in part the person taking the test, against the background of the paper, which represents his environment. The Greek Cross is shown, with instructions to copy, but not trace the figure on a standard sheet of paper. Abnormalities include failing to reproduce each arm of the cross, particularly the right arm, with a square form, which is separated from the other arms by 90 degrees. (Figure 1)

The Bender Visual Motor Test[2,13] (Figure 2) requires the subject to copy 9 standard cards, obtainable from the Psychological Corporation, and takes 5-10 minutes. While deciding on psychiatric treatment, neurosurgical intervention, or hospitalization, such time expenditure is relevant. Abnormalities in adults consist in misconception of the forms, and misunderstanding of the relationships between their parts.

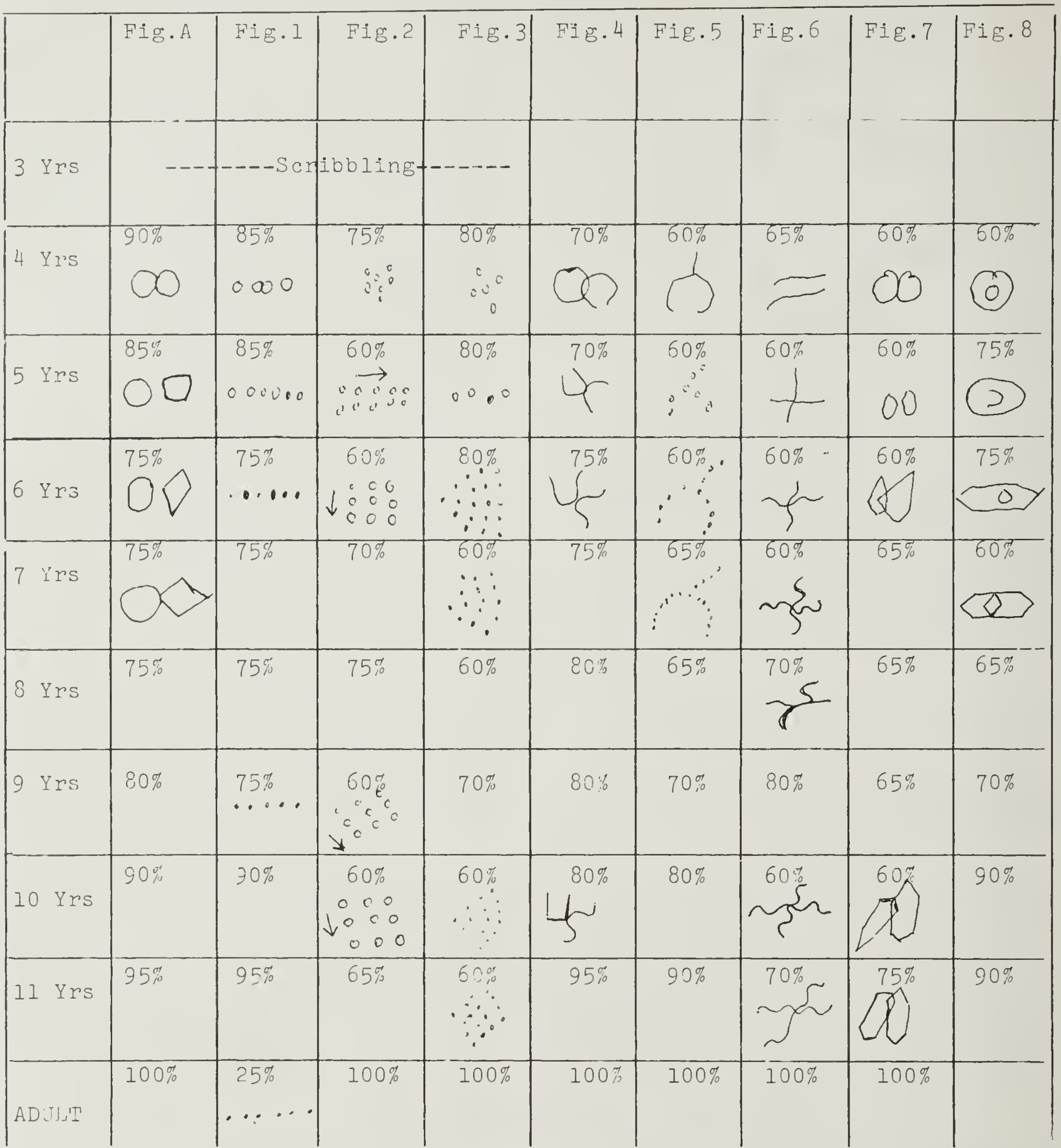


	Fig.A	Fig.1	Fig.2	Fig.3	Fig.4	Fig.5	Fig.6	Fig.7	Fig.8
3 Yrs	--------Scribbling-------								
4 Yrs	90%	85%	75%	80%	70%	60%	65%	60%	60%
5 Yrs	85%	85%	60%	80%	70%	60%	60%	60%	75%
6 Yrs	75%	75%	60%	80%	75%	60%	60%	60%	75%
7 Yrs	75%	75%	70%	60%	75%	65%	60%	65%	60%
8 Yrs	75%	75%	75%	60%	80%	65%	70%	65%	65%
9 Yrs	80%	75%	60%	70%	80%	70%	80%	65%	70%
10 Yrs	90%	90%	60%	60%	80%	80%	60%	60%	90%
11 Yrs	95%	95%	65%	60%	95%	90%	70%	75%	90%
ADULT	100%	25%	100%	100%	100%	100%	100%	100%	

Developmental Norms For The Bender Gestalt

Figure 3

Figure 3 shows the development of performance on this test by children's years of age.

Left Hemisphere

Two simple psychologic tests for lesions of the left cerebral hemisphere are the Serial Sevens and Wechsler Memory Scale. In the Serial Sevens, the subject is asked to "take away 7 from 99 (or 101, or 102, but not 100, as the senior author has examined a number of patients with brain syndromes who memorized the series beginning with 100), and to count downwards by sevens, giving me the answer for each count." Abnormality for adults of average intelligence consists of one error or more.

In the Wechsler Memory Scale, the subject is asked to start with the number 1 and count forward by threes, and is stopped at 40, which is the thirteenth correct answer. Abnormality for adults of average IQ consists of one error or more.

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PKU SCREENING IN ARIZONA:

A report of two (missed) cases and thoughts for future planning.

J. KIPP CHARLTON, M.D.

MARGARET S. TENBRINCK, M.D.

Is Legislation Needed to screen for PKU? These authors suggest a broader genetic disease screening program rather than a law for each disease, seemingly a wiser and more flexible approach. Critics of governmental meddling may find this difficult to swallow, but have alternatives been forthcoming?

INTRODUCTION

Two previously undiagnosed cases of retarded siblings with phenylketonuria in a Mexican-American family with five living children, have recently been identified at the Child Evaluation Center, Phoenix.

It is felt that these cases illustrate some of the problems with the present system of screening, diagnosis, and follow up of PKU.

CASE REPORTS

1. S. L. was born uneventfully at term on October 9, 1973 at a Phoenix hospital as the sixth child to a then 24 year old mother and 27 year old father. Her birth weight was 3.24 kg (7 lbs. 1 oz.), length 52 cm (20.5 in.), head circumference 34.5 cm and Apgar scores were nine at one and five minutes.

The phenylalanine level on the second day of life was 6.2 mgm%, but the report was not recorded in the chart. The hospital sent the report to the clinic where S. L. was to receive her well child care, but there is no record of such a report being received.

She was taken for check ups at 2, 3 and 5 weeks and at 5, 6, and 9 1 2 months. At 17 months of age she was referred to the Child Evaluation Center for evaluation of slow development.

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From: Department of Pediatrics, Maricopa County General, 2601 E Roosevelt, Phoenix, AZ 85008 (Dr. Charlton). Child Evaluation Center, Phoenix, AZ (Dr. Tenbrinck, Retired Director). Reprint requests to Dr. Charlton.

When seen there at 25 months of age her height was 84 cm (33 in.), 10-25th percentile, weight was 11.3 kg (25 lbs.), 25th percentile. The mother confirmed that S. L. sat at one year, walked at two years and never attempted to imitate sounds or language. Her hair was a light brown, very much lighter than the parents' brown-black hair. She was hypotonic and hyporeflexic.

Her hearing level was 50 dB in the right ear and 80 dB in the left. The EEG was mildly abnormal. Chromosome analysis revealed 46, XX 9qht+ (heteromorphic, clinical significance undetermined).

A urine amino acid screening test revealed an increase in phenylalanine. The serum phenylaline level was 31 mg%, and the tyrosine 1.0 mg% (normal 0.9-2.9).

2. A urine ferric chloride test on the other members of the family revealed that a brother J. H. L., age five, was the only other one with a positive test. He was born September 11, 1970 in Brigham, Utah. Birth weight 3 kg (6 lbs. 11 oz.), length 51.75 cm (20.5 in.), head circumference 33 cm (13 in.), and Apgar scores of ten at one and five minutes. Although a PKU test was performed on the second day, the results are not on the birth record, and the family returned to Arizona when he was two weeks old. He had been followed in the same facility as his sister.

When seen at the Child Evaluation Center the mother reported that he began walking at three years and talking at four years. His school (kindergarten) performance was estimated to be that of a three year old. On examination his height was 111 cm (43 in.), 25-50th percentile, weight 19.5 kg (42 3/4 lbs.), 50-75th percentile, and the head circumference was 51 cm, just below the 50th percentile. His hair was medium brown with some gray hairs over the crown.

Serum phenylalanine was 26.5 mg% and tyrosine 0.7mg% (normal 0.9-2.9).

DISCUSSION

It is too easy to point out how many Phoenix hospitals, clinics, and physicians missed the opportunity of making the proper diagnosis. The striking feature in the case of S. L. is that the diagnosis *was* made after birth, but the report was overlooked or misplaced. This highlights the weakness of our present voluntary, fragmented system of PKU screening. Both children were screened, but then "fell through the cracks".

The bottom line, we feel, is the need in Arizona for a state organized and funded mandatory screening program with adequate follow-up services, not only for PKU but for other genetic and/or treatable disease. In the discussion which follows, we will attempt to make the case for this conclusion.

There seems little doubt that from a monetary standpoint PKU screening is cost-effective. Komrower's[1] figures (if one can extrapolate from Manchester, England) indicates that one dollar spent for the screening and treatment of PKU is more than balanced by a savings of $4.48 in institutional and other care which would otherwise be given to non-screened, untreated, retarded PKU patients. Scriver[2] quotes similar data. Parenthetically, this kind of cost analysis is based on the incidence of PKU in a given population and may differ in Arizona. We have been unable to obtain frequency data for the Latin-American population but these cases clearly indicate that PKU does occur in this group.

All of this is not to say that the decision to screen is a monetary one exclusively. Thus, in the extreme, one could effect a considerable savings by eliminating chronic care institutions for the mentally retarded. More realistically, one must consider the ultimate social worth of the individual protected from disease; his earning power as an adult, the reduction of adverse psychologic and social effects on his family, the psychological rewards to health workers, etc.

While most infants in this country are screened on the third or fourth day of life,[3] in many hospitals multiparous mothers are discharged within 48 hours (as was patient S. L.) and infants of primiparous mothers with 72 hours. It is generally agreed, that in patients with classic PKU the blood levels of phenylalanine rise with time (in fact in males it is a linear relationship[4]). It is not surprising then that Starfield,[3] in data gathered from eight states, found 23 infants whose initial test results were negative but who were subsequently proved to have PKU (blood phenylalanine levels greater than 20 mg%). She calculates this as 8.3% missed by PKU screening.

There is great deal of evidence to support this notion. In the United Kingdom where the screening is done in the homes by "Health Visitors" on about the sixth to eighth day of life, no cases missed by screening could be found.[3] Using USA data, the incidence of PKU in infants screened before the fourth day of life is 5.2 per 100,000 screened, while after the fourth day of life the incidence is 8.4 per 100,000.[5] As mentioned earlier, many hospitals would have to accept an even higher "missed" rate due to the even earlier discharge, or we would have to organize an efficient, effective system for delayed screening.

Follow-up testing of infants with elevated screening tests is delayed in many states. Overall the interval between the initial and follow-up test is 25 days (vs. 12 days in the U.K.). There is evidence that infants treated within the first three weeks of life have a better outcome than those treated between three and six weeks of age. Thus, this delay may have some bearing on the prognosis.

Holtzman[5] points out that PKU screening in other states is less than optimal. Thus, for individual states the percentage of children screened varies from 62.3% to 100%. Each of the states investigated has legislation requiring PKU screening.

Holtzman also notes that the great variation in the incidence of PKU from state to state suggests that errors in the performance and interpretation of the Guthre test exist. This may be due to the large number of laboratories which are authorized to perform the test in the states where the "incidence" is low. The incidence is the highest where the test is only done in a few accredited labs. Limiting and or centralizing the number of laboratories doing the test should also simplify data collecting, statistical analysis and follow-up activity as well as being cost effective.

As mentioned earlier, the greatest impediment to our setting up a PKU screening program is the basic operating cost and the logistics of follow-up in a sometimes poorly informed, perhaps unmotivated population.

Scriver[2] develops the concept of what he calls "marginal cost". This is the cost for any additional test procedure in the same (PKU) program. This marginal cost comprises only the new materials and equipment required for the new test. Theoretically, this cost should be minimal since the same system of collecting specimens, recording necessary demographic data, transporting specimens, recording and reporting results, etc. can be used for the new test.

An instructive example of this is the PKU screening program in Manchester, England.[1] There, one-dimensional chromatography of plasma is performed because six aminoacidemias can be looked for simultaneoulsy. In the 206,288 babies screened from 1965 to 1973, 31 cases of PKU were detected; nine cases of histidinemia and 10 cases of hyperprolinemia were also found. The Province of Quebec includes blood tests for PKU, tyrosinemia and galactosemia and additional urine tests for 30 other metabolic disorder. In Massachusetts repeat screening is accomplished by a request to the mother to obtain and mail a urine specimen when the baby is three to four weeks old. The cost of this procedure is justified by the use of the specimen to screen for other conditions. These additional tests are particularly important for disorders like galactosemia and maple syrup urine disease since early treatment is imperative.

One might consider routine (even mandatory) testing of newborns for cystic fibrosis since it is (at least five to ten times) more common than PKU. We have recently seen a case of hypothyroidism that might have been detected in the newborn period if screening for this disorder were being performed as it is in Oregon.[6]

> K. B. was born at Maricopa County General Hospital on February 23, 1975 to a G2 P1 19 year old woman following an uncomplicated pregnancy, labor and delivery. The birth weight was 9 lb. 14 oz. (4494 gm) OFC 38.1 cm, and length 57.2 cm. She was discharged on February 26, 1975 after an uneventful three day stay. Specifically, the temperature was always 98°, stool was passed on the third day of life and feeding and activity were considered "normal".
>
> At seven months of age she was referred to Maricopa County General Hospital because of failure to thrive, lethergy, edema of the extremities, umbilical hernia, protruding tongue, poor muscle tone, poor appetite, constipation, "coarse" skin and hoarse cry. Her weight was now 13 lb. 9 oz. ($<3\%$), length 60 cm ($<3\%$), OFC 40 1 2 cm ($<3\%$). ETR was 0.72 (normal 0.86-1.13), T_3 30% (normal 34-45), T_4 1.2 μgm% (normal 4.5-11.5), TSH 50 μU ml (normal 1.5-5.5), and the bone age was delayed.
>
> She was responded slowly to thyroid replacement therapy.

Thus, it may well be that we should consider not just recommending (mandatory) PKU testing, but rather a program modeled after Quebec, Manchester, or Massachusetts.

Sadly, in gathering data from states that require PKU testing, Holtzman[5] asked six questions: only four states were keeping sufficient information to be able to answer them completely. In summary then, the low proportion of live births screened, the occurance of PKU infants missed by screening, the variation in

PKU "incidence" from state to state, and the long interval between screening and follow-up, seem to militate for something more than just another PKU screening program.

We would suggest an integrated, comprehensive, state-wide, centrally organized, genetic disease program; perhaps similar to that recommended by Holtzman in Maryland.[7,8] In this sceme a committee of experts ("Committee on Hereditary Disorder") decides what diseases should be screened for, rather than having the law itself name each test specifically. The Maryland law is now considered to be model legislation.

There are, of course, many other models that could be looked at, but most are perhaps unrealistic. As an example, the use of a "Health Visitor"[9] who might be required to visit the home of every child by two weeks of age, is particularly intriguing. This visit could be used to good advantage for purposes other than genetic screening—evaluating the potential for child abuse, for example.

There are many other issues that need to be discussed in developing legislation; confidentiality, retention of data, adequate genetic counseling, informed consent to name a few.

The purpose of this communication, however, is not to analyze specific legislation but rather to suggest that the tragic cases presented above should force us to reevaluate our present system (or non-system) of screening for PKU—and hopefully other diseases as well.

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HEAD AND NECK PAIN A DENTAL APPROACH

MICHAEL H. WEISS, D.D.S.

The patient with the complaint of head and neck pain is a frequent visitor to physicians. Usually, there is no gross abnormality found on examination. Commonly overlooked posterior causes of pain are lesions of the cervical spine.

Lateral causes of pain include the temporomandibular joints. As the author points out, it is easy to palpate these superficial joints for local tenderness and easy to look at the teeth to see if there is an equal bite on both sides. If teeth are missing, chewing is impaired, the TMJ function is impaired and poor nutrition results, also.

TMJ pain is not always over the location of the joint but may be referred to the side of the face or neck.

Those aching, boring, searing and lightning-like jabs that afflict people in the head and neck area often lead to a diagnostic maze as complex as the nerve pathways that conduct the pain. Unfortunately, they sometimes send the patient on an expensive, time-consuming and often futile search of health practitioners in an effort to find relief.

If the usual medical evaluation and treatment does not result in patient relief, the differential diagnosis should include a dental approach. The myofascial (MPD) and temporomandibular joint (TMJ) pain dysfunction problem is in the "grey area" or "no man's land" between dentistry and medicine.

The professional literature is now focusing on the problem of MPD syndrome, as evidenced by a recent questionnaire survey of physicians, dentists and dental educators in Canada and the United States.[1] It revealed widespread difference of opinion among both physicians and dentists on nearly every aspect of this syndrome. Of those surveyed, only 51 per cent of the physicians and 65 per cent of the dentists acknowledged the existence of TMJ dysfunction syndrome. How-

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Submitted for publication 6 76
From: 1158 E. Missouri Ave., Phoenix, AZ 85014. Reprint requests to Dr. Weiss.

ever, a considerable percentage of those sampled thought that a variety of functional disturbances and organic changes in the body could result from TMJ dysfunction problems.

The MPD and TMJ problem most often arises from excited or spastic musculature triggered by the mechanics of dental malocclusion.[2] If this is the underlying cause of the pain syndrome, it requires a bio-mechanical solution, rather than a drug therapy approach.[3]

The dental examination (Figures 1 and 2) should include a thorough history of the pain dysfunction, and among dentistry's diagnostic tools are TMJ x-rays, study dental casts, electronic mandibular tracing devices,[4] palpation of craniomandibular musculature, (including the masseter m., temporalis m., and internal pterygoid m. which close the madible; the external pterygoid m., and digastric m. that open the mandible), measurement of the amount of mouth opening, mandibular deviations, auscultation of the TMJ and transcutaneous electronic stimulation.[5] The orthopedically oriented dentist, by careful evaluation of the preceding facts, can determine whether or not the patient's head and neck pain arises from a dental cause.

The physician could suspect a problem of head and neck pain of dental origin (jaw imbalance) by asking the patient the following questions:

1. Did the onset of pain come about following tooth removal, dental treatment or general trauma of the TMJ area?
2. Can the individual open his mouth adequately, chew properly or swallow without discomfort?
3. Is there pain or are there clicking noises in the hinges in front of the ears?
4. Is the patient aware of clamping or grinding his teeth?
5. Are the jaw muscles tender, especially upon awakening?

Very simple office tests can be performed, such as palpating the temporomandibular joint by either placing the small fingers in the ears, or pressing the area immediately in front of the tragus of both ears while having the patient open and close his mouth. If painful responses are elicited, we can reasonably assume that joint derangement is present.

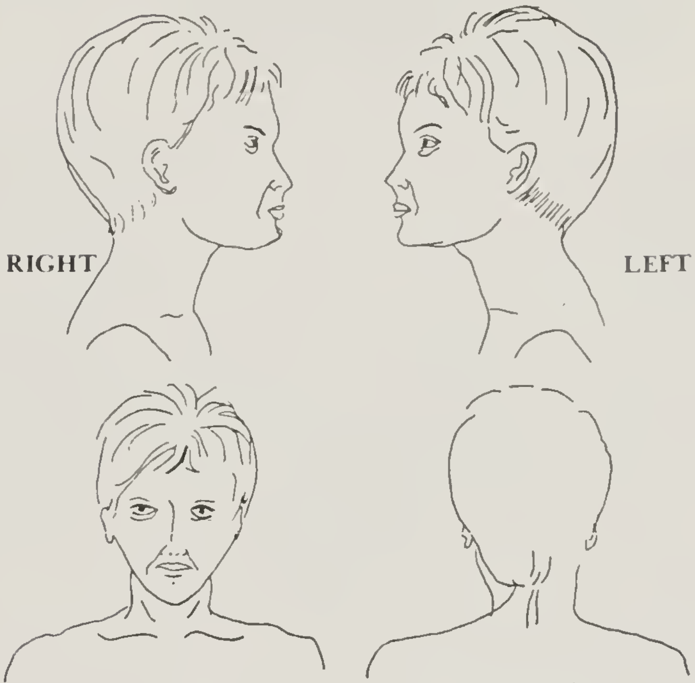
Dental signs can be noted by the extreme wear of the teeth or deviation from side to side (erratic movement) of the lower jaw in slowly opening and closing the mouth.

These are some of the subjective and objective signs that the physician can note in his differential diagnosis. If they are present, the patient should be referred to a dentist who is trained in the treatment of the myofascial pain dysfunction syndrome.

DENTAL EXAMINATION

Myo-Fascial and
Temporo-Mand. Joint
Pain and Dysfunction

DOT IN AREAS OF PAIN



CHIEF COMPLAINT—
ONSET OF PAIN—
DURATION OF PAIN—
SIDE OF PAIN— RIGHT— LEFT—
BILATERAL—
TYPE OF PAIN—
PREVIOUS DENTAL TREATMENT—
PREVIOUS MEDICAL TREATMENT—
COMMENTS—

Figure 1

Clinical experience has demonstrated that dysfunction of the dental occlusion, temporomandibular joint and associated neuromusculature is a prime factor in the chain of events which begins with a dental malocclusion and terminates in a variety of types of facial and head discomforts. (Figure 3) The involved neuromusculature is composed of the sensory and motor branches of the trigeminal nerve (cranial V) and facial nerve (VII) that supply the muscles of the head and neck. Some or all of the following symptoms which include head and neck pains, "Headaches", tinitus, selected types

DENTAL EXAMINATION

Name ____________________________________

Date ____________________

1. Do you ever wake in the morning with aching jaws? Or painful teeth?
2. Do you have difficulty in opening and closing the mouth?
3. Do you have any facial or neck pain?
4. Do you have any pain, clicking or popping sounds in front of your ear?
5. Do you ever have stuffiness or ringing in your ears?
6. Do you have headaches?
7. Are you aware of clamping or grinding your teeth?
8. Do you have discomfort with the use of cold or sweets?
9. Is there any discomfort in mastication?
10. Are you a nervous or tense individual?
11. Do you bite your tongue or cheeks?
12. Sleep problem?
13. Medication—tranquilizer or muscle relaxant, or drugs for pain?

Degree of Pain in Palpation			**Objective Symptoms**
1. Trapezius M.	R.	L.	1. Max. opening of jaw in m.m.
2. Sterno C. Mast. M.	R.	L.	2. Freeway space in m.m.
3. Ext. Ptery M.	R.	L.	3. Deviation of jaw in opening
4. Int. Ptery M.	R.	L.	4. Ausc. of T.M.J.
5. Mass M.	R.	L.	5. Degree of tooth mobility Class I, II, III, IV
6. Temporal M.	R.	L.	6. Bruxism—degree of tooth destruction
7. T.M.J.	R.	L.	7. T.M.J. X-Ray findings:
			8. Periapical X-Ray findings:

Figure 2

of hearing loss (conductive), vertigo, earaches, clicking and crepitus in the temporomandibular joint, limited mandibular movement, pain in shoulder, difficulty in swallowing and a host of other bizarre complaints, may be apparent on history and examination.[6]

Normal dental occlusion occurs over 1,800 times daily immediately preceding deglutition. This act requires the craniomandibular musculature to forcefully join the maxillary and mandibular dentition in order to generate the forces required to swallow. If a malocclusion exists, more muscle tension must be applied to this bony junction to achieve articular stability.

The consistent pattern of muscle tension produces ischemia, trismus and localized edema.[7] In a patient with atonic or spastic facial musculature, the localized metabolic controls are not able to function in their normal manner. The resulting accumulation of metabolites leads to osmolarity changes that eventually show clinically detectable facial edema.

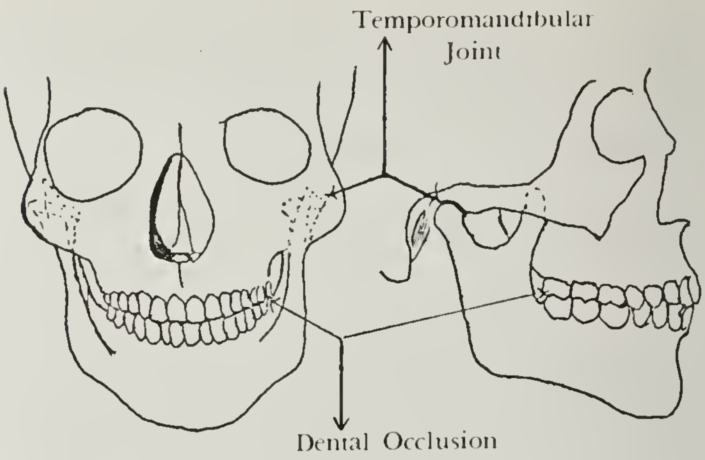


Figure 3

Location of the anterior and lateral views of the temporomandibular joint and dental occlusion.

As a result of this misdirected muscle activity, much damage is seen by the trained dentist. For example: Abnormal attrition of teeth, breakdown of the peridontium, loss of dental alveolar bone, and changes in the temporomandibular joint may occur. When the patient cannot cope with the muscle pathology, the typical subjective symptoms of myofascial pain dysfunction syndrome appear clinically.

Sound dental treatment is predicated on the relief of muscle malfunction by eliminating the underlying causes of the dental malocclusion. Treatment could include dental coronaplasty, plastic splints, restorations and prosthesis designed to reorient the jaw to normal function.

It does not occur to most patients to complain of chronic headache to their dentist, and most physicians are not attuned to looking for a dental malfunction as a cause of head and neck pain. If the physician and dentist both recognize the etiology, they can orient their treatment of head and neck pain towards an inter-relationship that will benefit their patient.

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hen you control urine volume in your patients...

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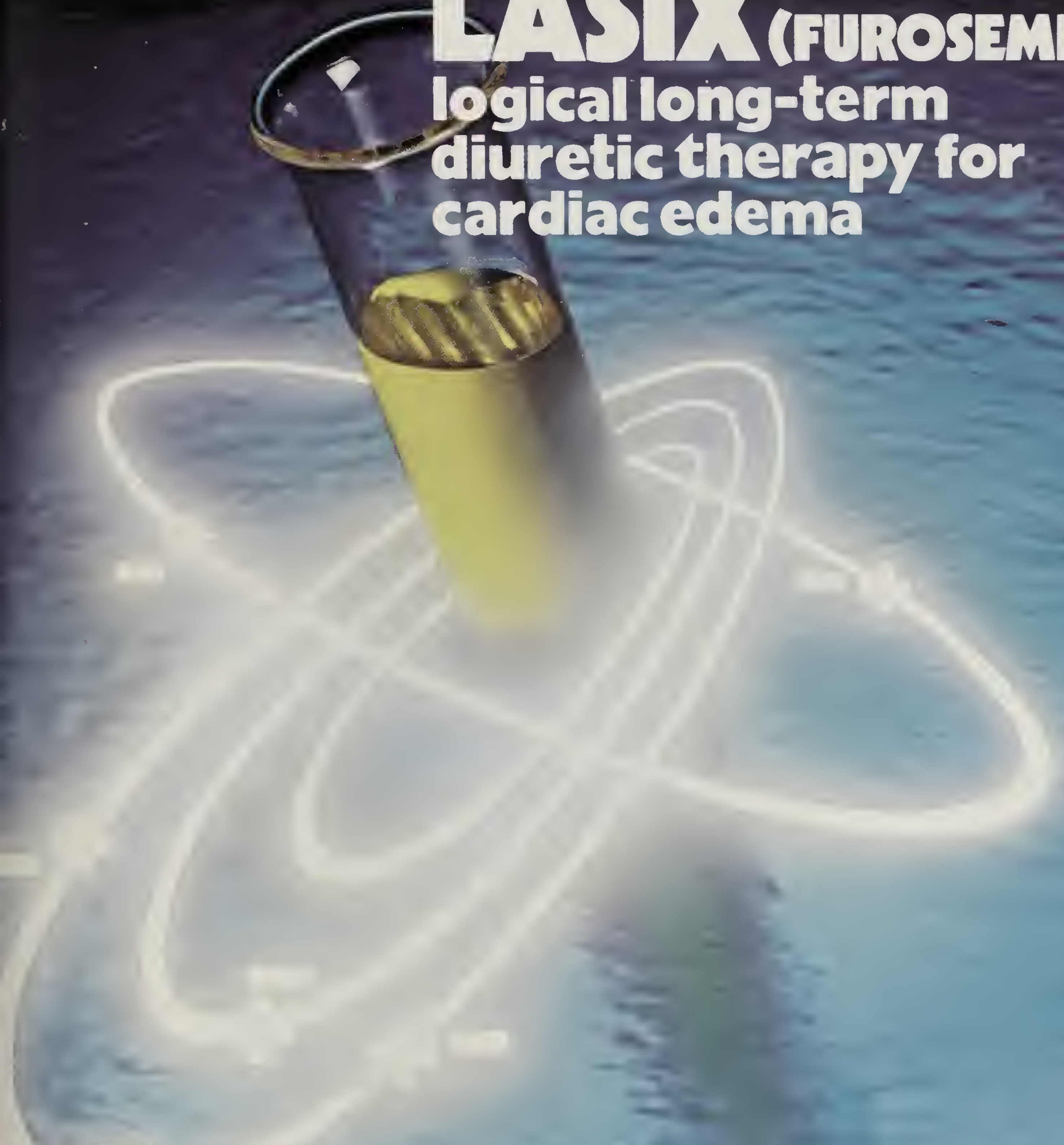
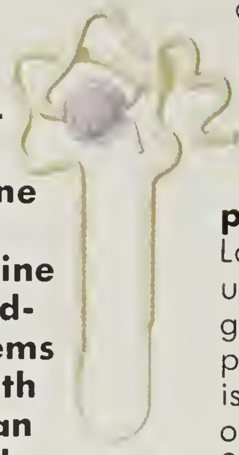
logical long-term diuretic therapy for cardiac edema



(Please see last page of this advertisement for prescribing information.)



The Lasix® site of action is a distinctive advantage—for it results in fewer electrolytes in each liter of urine than with other commonly used diuretics.[1,2] That is why, if you control urine volume through individual dosage adjustment, you may face fewer problems with electrolyte disturbances.[3] As with any diuretic, electrolyte depletion can occur during therapy with furosemide. Careful clinical and laboratory observations are advisable.

The primary site of Lasix® action—at the loop of Henle—distinguishes Lasix from other commonly used diuretics. The action at this site enables Lasix to produce a dilute urine,[4] which may be the basis for its relative freedom from electrolyte disturbances (hypokalemia).[3] When the edematous patient has been stabilized, excessive diuresis can usually be avoided by simple dosage adjustment. And, through regulation of urine volume by this dosa adjustment, you can control electrolyte excretio. Careful medical supervision is required.

Wide dose-related effectiveness for precise control of diuretic response. The Lasix® effect is dose-related. A prompt diuresis usually ensues with a single dose of 20 to 80 mg, given preferably in the morning. Depending on th patient's response, a second dose may be administered 6 to 8 hours later. Clinical and laboratory observations are advisable when doses exceedin 80 mg/day are given for prolonged periods. Whe maintenance of the edema-free state is desired, dosage may be adjusted to as low as 20 mg.

(Please see the dosage and administration section on the last page of this advertisement.)

Highly effective/short acting. The effectiveness of Lasix® has been clearly establishe in hundreds of thousands of patients in a decade clinical experience. Consequently, Lasix is the most selected diuretic agent today in the treatme of edemas associated with congestive heart failu

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LASIX® (FUROSEMIDE)

the diuretic for both initial & maintenance therapy in cardiac edema

The onset of diuresis usually occurs within an our and is usually complete within 6 to 8 hours; hus, patient inconvenience is minimized.

Useful in patients with renal impairment and severely depressed renal function. Unlike other commonly used diuretics, Lasix® an be useful in patients with renal impairment nd severely depressed renal function. Even in the resence of renal insufficiency with depressed lomerular filtration, acidosis, alkalosis or hypolbuminemia, Lasix has been found effective and well tolerated.[5] If increasing azotemia and oliguria ccur during treatment of severe progressive renal disease, the drug should be discontinued.

(See next page for prescribing information.)

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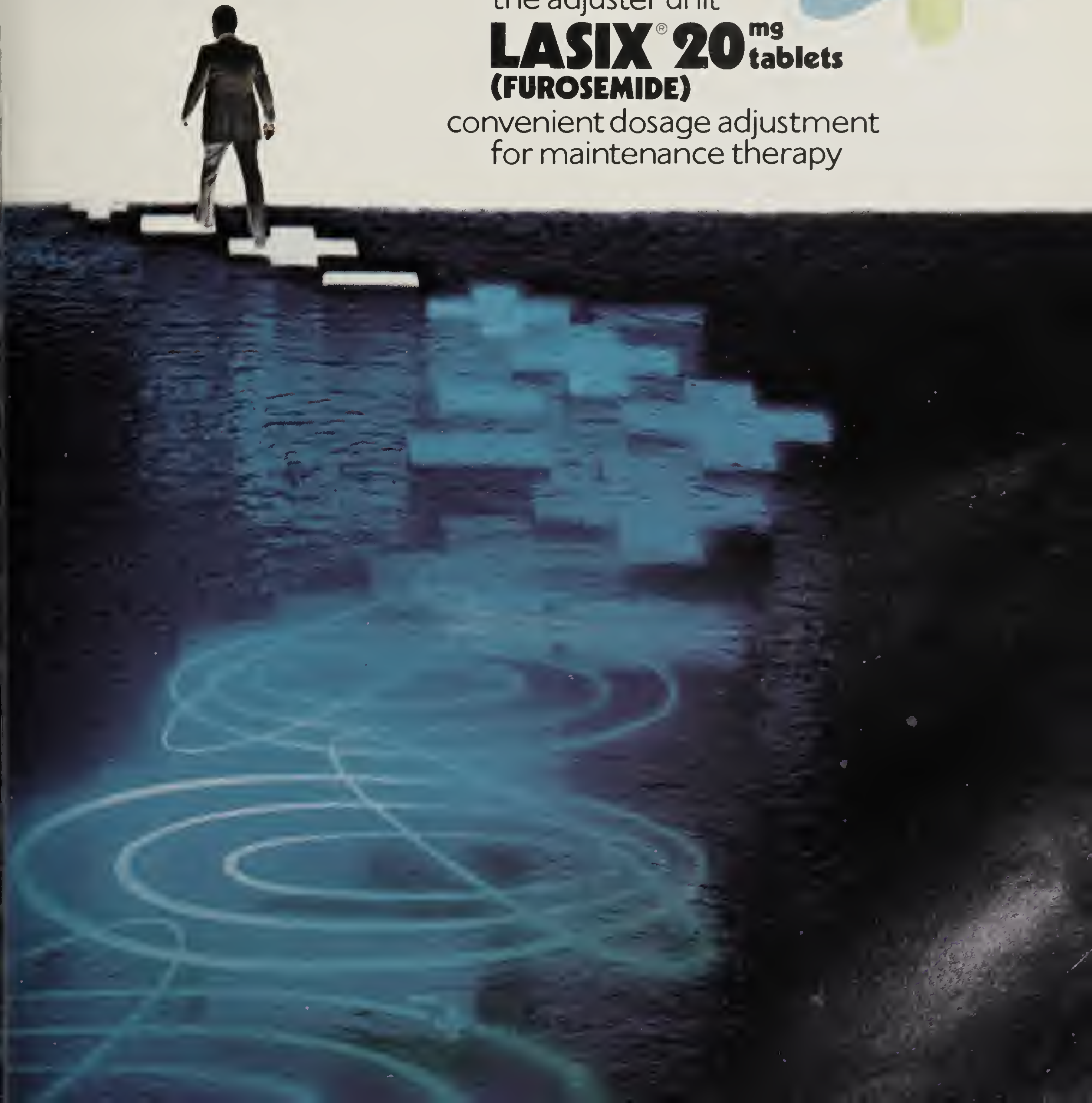

LASIX® 40 mg and 20 mg tablets. (FUROSEMIDE)

logical long-term diuretic therapy for cardiac edema

A brief summary of the Prescribing Information for

Lasix® (furosemide) Tablets 20 mg and 40 mg

WARNING—Lasix (furosemide) is a potent diuretic which if given in excessive amounts can lead to a profound diuresis with water and electrolyte depletion. Therefore, careful medical supervision is required, and dose and dose schedule have to be adjusted to the individual patient's needs. (See under "Dosage and Administration.")

Indications—Lasix (furosemide) is indicated for the treatment of the edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Hypertension—Lasix (furosemide) may be used for the treatment of hypertension alone or in combination with other antihypertensive drugs. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controllable with Lasix (furosemide) alone.

CONTRAINDICATIONS—Because animal reproductive studies have shown that Lasix (furosemide) may cause fetal abnormalities, the drug is contraindicated in women of childbearing potential. (See "Additional Information.")

Lasix (furosemide) is contraindicated in anuria. If increasing azotemia and oliguria occur during treatment of severe progressive renal disease, the drug should be discontinued. In hepatic coma and in states of electrolyte depletion, therapy should not be instituted until the basic condition is improved or corrected. Lasix (furosemide) is contraindicated in patients with a history of hypersensitivity to this compound.

Warnings—Excessive diuresis may result in dehydration and reduction in blood volume, with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients. Excessive loss of potassium in patients receiving digitalis glycosides may precipitate digitalis toxicity. Care should also be exercised in patients receiving potassium depleting steroids.

Frequent serum electrolyte, CO_2 and BUN determinations should be performed during the first few months of therapy and periodically thereafter, and abnormalities corrected or the drug temporarily withdrawn.

In patients with hepatic cirrhosis and ascites, initiation of therapy with Lasix (furosemide) is best carried out in the hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma; therefore, strict observation is necessary during the period of diuresis. Supplemental potassium chloride and, if required, an aldosterone antagonist are helpful in preventing hypokalemia and metabolic alkalosis.

Patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

In those instances where potassium supplementation is required, an oral liquid preparation should be used rather than enteric-coated potassium salts.

There have been several reports, published and unpublished, concerning nonspecific small-bowel lesions consisting of stenosis, with or without ulceration, associated with the administration of enteric-coated thiazides with potassium salts. These lesions may occur with enteric-coated potassium tablets alone or when they are used with nonenteric-coated thiazides, or certain other oral diuretics.

These small-bowel lesions have caused obstruction, hemorrhage, and perforation. Surgery was frequently required, and deaths have occurred.

Available information tends to implicate enteric-coated potassium salts, although lesions of this type also occur spontaneously. Therefore, coated potassium-containing formulations should be administered only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Patients with known sulfonamide sensitivity may show allergic reactions to Lasix (furosemide).

Precautions—As with any potent diuretic, electrolyte depletion may occur during therapy with Lasix (furosemide), especially in patients receiving higher doses and a restricted salt intake. Electrolyte depletion may manifest itself by weakness, dizziness, lethargy, leg cramps, anorexia, vomiting, and/or mental confusion.

Asymptomatic hyperuricemia can occur and gout may rarely be precipitated. Reversible elevations of BUN may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

When parenteral use of Lasix (furosemide) precedes its oral use, it should be kept in mind that cases of tinnitus and reversible hearing impairment have been reported. There have also been some reports of cases in which irreversible hearing impairment occurred. Usually, ototoxicity has been reported when Lasix (furosemide) was injected rapidly in patients with severe impairment of renal function at doses exceeding several times the usual recommended dose and in whom other drugs known to be ototoxic were often given. If the physician elects to use high dose parenteral therapy in patients with severely impaired renal function, controlled intravenous infusion is advisable (for adults, it has been reported that an infusion rate not exceeding 4 mg Lasix [furosemide] per minute has been used).

Increases in blood glucose, and alterations in glucose tolerance tests with abnormalities of the fasting and two-hour postprandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Lasix (furosemide) may lower serum calcium levels, and rare cases of tetany have been reported.

Patients receiving high doses of salicylates, in conjunction with Lasix (furosemide) may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Diuretics such as furosemide may enhance the nephrotoxicity of cephaloridine. Therefore, Lasix (furosemide) and cephaloridine should not be administered simultaneously.

Sulfonamide diuretics have been reported to decrease arterial responsiveness to pressor amines and to enhance the effect of tubocurarine. Great caution should be exercised in administering curare or its derivatives to patients undergoing therapy with Lasix (furosemide), and it is advisable to discontinue Lasix (furosemide) for one week prior to any elective surgery.

Adverse Reactions—Various forms of dermatitis, including urticaria and rare forms of exfoliative dermatitis, erythema multiforme, pruritus, paresthesia, blurring of vision, postural hypotension, nausea, vomiting, or diarrhea.

Anemia, leukopenia, aplastic anemia, and thrombocytopenia (with purpura). Rare cases of agranulocytosis which responded to treatment.

In addition, the following rare adverse reactions have been reported; however, relationship to the drug has not been established with certainty: sweet taste, oral and gastric burning, paradoxical swelling, headache, jaundice, thrombophlebitis and emboli and acute pancreatitis.

Lasix (furosemide)-induced diuresis may be accompanied by weakness, fatigue, lightheadedness or dizziness, muscle cramps, thirst, increased perspiration, urinary bladder spasm, and symptoms of urinary frequency.

Dosage and Administration

ADULTS

The usual adult dose of Lasix (furosemide) is 20 to 80 mg given as a single dose.

If the diuretic response with a single dose of 20 to 80 mg is not satisfactory, the following schedule should be used: Increase this dose by increments of 20 or 40 mg not sooner than 6 to 8 hours after the previous dose until the desired diuretic effect has been obtained. This individually determined single dose should then be given once or twice daily. The dose of Lasix (furosemide) may be carefully titrated up to 600 mg per day in those patients with severe clinical edematous states.

With doses exceeding 80 mg/day and given for prolonged periods, careful clinical and laboratory observations are particularly advisable.

Hypertension—The usual dose of Lasix (furosemide) is 40 mg twice daily both for initiation of therapy and for maintenance. Careful observations for changes in blood pressure must be made when this compound is used with other antihypertensive drugs, especially during initial therapy. The dosage of other agents must be reduced by at least 50 percent as soon as Lasix (furosemide) is added to the regimen to prevent excessive drop in blood pressure. As the blood pressure falls under the potentiating effect of Lasix (furosemide), a further reduction in dosage, or even discontinuation, of other antihypertensive drugs may be necessary. It is further recommended, if 40 mg twice daily does not lead to a clinically satisfactory response, to add other hypotensive agents, e.g., reserpine, rather than to increase the dose of Lasix (furosemide).

INFANTS AND CHILDREN

Pediatric Administration: The usual initial dose of oral Lasix in infants and children is 2 mg/kg body weight, given as a single dose. If the diuretic response is not satisfactory after the initial dose, dosage may be increased by 1 or 2 mg/kg not sooner than 6 to 8 hours after the previous dose. Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

How Supplied—Lasix Tablets 40 mg (furosemide) supplied as white, round, monogrammed, scored tablets.

Lasix Tablets 20 mg (furosemide) supplied as white, oval, monogrammed tablets.

Note: Dispense in dark containers. Exposure to light may cause slight discoloration which, however, does not alter potency.

Additional Information

Toxicology

The acute toxicity of Lasix (furosemide) has been determined in mice, rats, and dogs. In all three animal species, the oral LD_{50} of Lasix (furosemide) exceeded 1000 mg/kg of body weight, while the intravenous LD_{50} ranged from 300 to 680 mg/kg. Intragastric injection of the drug in newborn rats resulted in an LD_{50} of 380 mg/kg.

The acute toxicity of high doses of Lasix (furosemide) was characterized by convulsions, paralysis, and collapse. Surviving animals often became dehydrated and depleted of electrolytes due to the diuresis induced by Lasix (furosemide). In the newborn rats, intragastric injection of the drug caused hyperactivity and anorexia.

Chronic toxicity studies with Lasix (furosemide) were done in rats and dogs. In a one-year study in rats, renal tubular degeneration occurred, with all doses higher than 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day). A six-month study in dogs revealed calcification and scarring of the renal parenchyma at all doses above 10 mg/kg (83 percent of the maximal recommended human dose of 600 mg per day).

Reproductive Studies

The effects of Lasix (furosemide) on embryonic and fetal development and on pregnant dams were studied in mice, rats, and rabbits.

Lasix (furosemide) caused unexplained maternal deaths and abortions in the rabbit when 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day) was administered between days 12 to 17 of gestation. In a previous study the lowest dose of only 25 mg/kg (2 times the maximal recommended human dose of 600 mg per day) caused maternal deaths and abortions. In a third study, none of the pregnant rabbits survived a dose of 100 mg/kg. Data from the above studies indicate fetal lethality which can precede maternal deaths.

The results of the mouse study and one of the three rabbit studies also showed an increased incidence of hydronephrosis (distention of the renal pelvis and, in some cases, of the ureters) in fetuses derived from treated dams as compared to the incidence in fetuses from the control group.

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THE MAN WITH THE MUCKRAKE

JOHN W. KENNEDY, M.D.

"Every one who has read Bunyan's Pilgrims Progress will remember the man with the Muckrake, the man who could look no ways but downwards with the Muckrake in his hands." Thus began Teddy Roosevelt in one of his excoriations of the "Yellow Journalists" of his day.

The names change but the problems remain the same. Now the Muckrakers will supply 'Expert Witnesses' to assist in any malpractice suit filed against you, and to testify for the plaintiff in his or her suit against you.

For the past several years, rumors have been floating up and down hospital corridors that there were 'finders' floating about hospitals. These are sometimes said to be hospital employees. It is said they contact patients and suggest that they secure the services of a legal nature to pursue a possible malpractice action. The same finders will direct the patient to the proper legal office. Not so long ago a patient indicated to me he had been so approached. In my abysmal ignorance I dismissed this as impossible and preposterous. How naive can one be.

It is now reported, "that physician-run organizations have been offering case analysis as a service to lawyers, and further reported that a physician run organization will analyse medical records for malpractice."[1] The solicitation is made to patients and finders.

The man with the Muckrake has gone full circle, you and I are the center of his undivided attention.

Full details of the 'finders fees', recruitment of 'finders' etc. is detailed in the article referred to—it is not a pleasant bedtime story.[1]

So we must practice, "preventive maintenance'. A place to start is that of office and hospital records. Yes, records are the bane of our existence but the hospital record committee, made up of your peers, may be one of the best "protective agencies" we have. When they ask that you get your records in order—they are assisting you and not hindering your practice of good medicine, and good malpractice 'preventive maintenance'.

---

[1] Medical World News, Oct 4, 1976.

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Dean's Page

GASCHROMATOGRAPH MASS SPECTROMETER

NEAL A. VANSELOW, M.D.

In January of this year a new facility, the Biomedical Mass Spectrometry Laboratory, was added to the College of Medicine. The Biomedical Mass Spectrometry Laboratory services are available to clinicians and researchers from within the College of Medicine and anyone else in the State of Arizona working in the fields of bioanalysis, toxicology or criminology, to mention just a few relevant areas. The facility is set up to be self-supporting and will charge users fees. A committee was appointed to oversee the operations of the Center and a very competent operator has been hired.

The new Biomedical Mass Spectrometry Laboratory is unique among other laboratories with similar instrumentation in its devotion to biomedical applications.

The main piece of equipment in the Laboratory is a Finnigan 3000 Gaschromatograph Mass Spectrometer with attached data handling computer. This instrument permits separation of materials extracted from physiological fluids if they are volatile or can be volatilized by chemical derivation in a gas chromatograph. The separated compunds then enter the mass spectrometer where an electron beam in a high vacuum causes the formation of compound-specific charged fragments which are then accelerated in the direction of the detector, separated according to their masses in a quadrupole mass sorter and detected very sensitively in the electron multiplier.

This instrument is a powerful analytical tool capable of detecting minute amounts of drugs and toxic materials in blood plasma, urine, or other physiological fluids. Nanograms (10^{-9} g) can often be identified and measured in a small volume of plasma.

The data handling computer allows for the reduction of the otherwise enormous amount of primary data to a more manageable size and, in addition, permits comparison of these data with a stored library of standards.

In this fashion trace amounts of unknown materials in a patient's blood plasma may be measured and the identity of the material determined by comparing the "fingerprint" of mass fragments the material produces in the instrument with fingerprints of known drugs and toxic materials sorted in the computer's memory.

As the Laboratory becomes fully active, it is hoped to acquire additional equipment which will allow the interchange via magnetic tape of data on drugs and toxic materials and their breakdown products generated here at the University of Arizona Health Science Center with those of other similar laboratories throughout the Nation. Another expansion intended at a future time is hookup of the Laboratory computer with a national computer library which will greatly enhance "search and match" capabilities.

We are very enthusiastic about the capabilities of this new instrument available to us. It will aid in ongoing and future research and clinical activities here at the Health Science Center. We also hope that we will be able to help interested parties on main campus and outside the University in their drug-related analytical problems.

To obtain information on details about using the equipment, one should contact Dr. Sai Chang (mass spectrometrist — 882-6011) or Dr. Klaus Brendel (Chairman, Users Committee — 882-7729).

Neal A. Vanselow

Clinical Oncology
In Arizona

ONCOLOGIC EMERGENCIES

STEPHEN E. JONES, M.D.

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Relatively few neoplastic conditions require immediate anti-neoplastic therapy. However, there is a group of conditions associated with malignancy which require immediate recognition and treatment to minimize morbidity or even premature mortality.[1] These entities may develop rapidly or insidiously and will be encountered at some time by every physician who cares for patients with malignancy. Some of these disorders are now being seen at a greater than expected frequency because treatment of the underlying tumor has improved and patients are surviving longer. For example, late central nervous system involvement by histiocytic lymphoma (spinal cord compression, diffuse leptomeningeal disease) is being observed more often now since a high proportion of patients with this type of lymphoma achieve prolonged remissions with chemotherapy.

Many patients with one of these oncologic emergencies will first be seen by their family or primary care physician. The purpose of this month's column is to briefly review the conditions which are true emergencies, the setting in which they occur, their more common clinical features, and their treatment.

I. Metabolic Emergencies

A. Hypercalcemia

This is one of the most commonly encountered oncologic emergencies. Hypercalcemia is generally seen in association with metastatic breast cancer, lung cancer, prostatic cancer, or multiple myeloma, but it can be seen in association with any type of cancer. The cause is usually bone metastases, but certain types of tumors secrete polypeptides which are responsible for hypercalcemia (parathormone-like activity produced by certain oat cell carcinomas of the lung, prostaglandin-like activity produced by certain prostatic cancers, etc.). The usual symptoms include changes in mental status, drowsiness, polyuria, thirst, constipation, anorexia, nausea or vomiting, weakness, or, rarely, a metallic taste in the mouth. The electrocardiogram may show a shortened QT interval. With severe hypercalcemia, sudden death due to cardiac arrythmias or asystole may occur. Prompt diagnosis and prompt treatment of hypercalcemia are critical to avoid an unfortunate outcome of a syndrome which is usually reversible. Many patients can be successfully managed for their underlying neoplasm for months or even years once the calcium is normalized.

Treatment for hypercalcemia consists of a number of therapeutic maneuvers. Mild hypercalcemia may be successfully managed with corticosteroids (prednisone, 60-80 mg day), a restricted calcium diet, increased fluids, and perhaps oral phosphates (for example, Fleet phosphosoda). Severe cases of hypercalcemia (>15 mg 100 ml) represent more of an emergency and should probably be managed with a variety of measures designed to normalize calcium rapidly, including forced hydration, diuretics, prednisone, and mithramycin. There is probably no role for the use of intravenous sulfates or phosphates as these may induce metastatic calcifications.

B. Hyperuricemia

Severe hyperuricemia may lead to acute uric acid nephropathy and renal shutdown. This is generally an avoidable complication. Patients who present with lymphoma or leukemia often are able to handle their uric acid loads until treatment is initiated. Treatment may precipitate marked cell destruction with a sudden increase in uric acid load. For newly diagnosed patients with leukemia or lymphoma, prophylactic therapy to prevent hyperuricemia and uric acid nephropathy should be initiated with allopurinol (300-

600 mg daily for several days) prior to the start of treatment. Other measures include careful observation of renal function, increased hydration, and, occasionally, alkalinization of the urine. If the patient does develop acute uric acid nephropathy, hemodialysis is an effective means of carrying him through this episode, often with a reasonable return of renal function. For most patients with lymphoma or leukemia, once treatment has been under way for approximately a month, allopurinol is no longer necessary. However, if the patient should relapse or again have bulky disease with a high cell loss factor, prophylactic therapy with allopurinol should again be instituted.

C. Hyperkalemia

Acute hyperkalemia is a rare complication of treatment with chemotherapy in a few patients with undifferentiated or poorly differentiated lymphomas. For example, in one study approximately 20% of patients with American Burkitt's lymphoma (diffuse, undifferentiated lymphoma) developed severe life-threatening hyperkalemia.[2] This emergency is probably due to rapid lysis of cells with resultant release of intracellular potassium. It may accompany hyperuricemia and strict attention to renal function, hydration, control of hyperuricemia, and monitoring of serum electrolytes is mandatory during the first course of chemotherapy for patients with bulky, undifferentiated types of lymphoma. Cardiac arrythmias, renal failure, or death can result from this complication if it is unrecognized.

II. Emergencies Directly Due to Tumor

A. Acute Leukemia

Acute leukemia, essentially regardless of cell type, is one of the few malignant diseases that requires immediate diagnosis and treatment. These patients often present with severe granulocytopenia and or thrombocytopenia because of extensive bone marrow infiltration by leukemia. These latter complications do not abate until a complete clinical and bone marrow remission has been achieved through the use of intensive, multi-drug, anti-leukemic therapy. This type of treatment generally necessitates a four- to -six-week period of hospitalization, prolonged bone marrow aplasia prior to recovery, and intensive supportive care with antibiotics, white blood cell transfusions, platelet transfusions, etc. Thus, there is no reason to defer treatment because that only prolongs the period of risk of infection or bleeding. Occasionally, older patients with acute leukemia will have a "smoldering" form of leukemia which is characterized by a low growth fraction, relatively indolent nature, and only slow progression with time. These patients often can be managed conservatively. In general, however, acute leukemia requires immediate diagnosis and treatment to minimize morbidity.

B. Airway Obstruction

This condition may be due to direct tumor growth into upper air passages (exophytic head and neck tumor, thyroid carcinoma, lung cancer, etc.), external compression of the airways by a large mediastinal mass, or recurrent laryngeal nerve involvement resulting in vocal cord paralysis. In many instances, the underlying condition is quite treatable and emergency control of the airway is necessary to provide sufficient time for treatment of the underlying condition. Treatment of airway obstruction might include tracheostomy or intubation, and emergency radiotherapy or emergency chemotherapy depending upon the underlying condition.

C. Brain Metastases

Any type of cancer can metastasize to the brain, but this condition is most frequently encountered in patients with breast cancer, lung cancer (up to 40% of patients with oat cell carcinoma of the lung) and malignant melanoma. Since the brain is a "privileged" site and since most cancer chemotherapeutic agents do not cross the "blood-brain barrier", brain metastases may develop at a time when other metastastic tumor is under adequate control. For example, in our program of combination chemotherapy of breast cancer with adriamycin and cyclophosphamide, 10% of our patients whose disease was responding peripherally had the first evidence of relapse in the central nervous system.[3] Brain metastases should be suspected in patients with known cancer who undergo a change in mental state or who develop new or unexplained neurologic findings. Occasionally, patients present with a stroke-like picture which on subsequent examination is found to be due to brain metastases from an underlying neoplasm. Emergency radionuclide brain scanning and or computed tomographic scanning will usually provide the diagnosis. Except in rare instances where the central nervous system metastasis might be solitary (for example, malignant melanoma, renal cell carcinoma), the treatment of choice is whole-brain irradiation.[4] In general, high-dose corticosteroid treatment (Dexamethasone, 16-24 mg daily) should be initiated once the diagnosis is suspected. At times, the use of corticosteroids results in objective improvement even before the initiation of whole-brain radiotherapy. Prompt diagnosis and prompt treatment will minimize residual neurologic dysfunction in these patients.

D. Spinal Cord Compression

These syndromes may occur insidiously or rapidly. Certain types of tumors are more prone to develop this complication: breast cancer, lung cancer, Hodgkin's disease, non-Hodgkin's lymphoma, multiple myeloma, prostatic cancer, and, rarely, leukemia ("chloromas" which are focal extradural collections of myeloblasts). The usual clinical manifestations are back pain associated with leg weakness and/or sensory changes. However, the patient may have all or none of these symptoms or may present with problems of bladder or bowel function instead. If the diagnosis is suspected, immediate neurological or neurosurgical consultation should be obtained and an emergency myelogram performed. If the diagnosis is thus confirmed, immediate neurosurgical decompression or emergency radiation therapy will control symptoms in most cases. The decision to use surgical decompression versus radiation therapy should be made in consultation. Often in patients with hematologic malignancies extremely close neurologic monitoring while emergency radiotherapy is being administered will be sufficient. However, in other patients where the changes are rapid or the tumors are not likely to be particularly radiosensitive (for example, malignant melanoma), the treatment of choice may be surgical intervention. Prompt diagnosis followed by immediate treatment will minimize neurologic deficits in these patients.

E. Diffuse Leptomeningeal Disease

Diffuse involvement of the leptomeninges by malignant cells is an uncommon variant of nervous system involvement by cancer. However, it is the most frequent type of involvement in certain disease states: acute leukemia, and diffuse histologic types of non-Hodgkin's lymphoma (particularly diffuse histiocytic lymphoma and Burkitt's lymphoma). Cranial nerve palsies, syndromes resembling spinal cord compression states, and vague neurologic symptoms in high-risk patients all are indications for examination of the spinal fluid. A cytocentrifuge preparation of spinal fluid stained with Wright-Giemsa stain will generally provide the diagnosis. The treatment is intrathecal administration of anti-cancer drugs (either cytosine arabinoside or methotrexate) and, in some cases, whole-brain irradiation as well. In childhood lymphocytic leukemia it is often necessary to place an Omaya reservoir for repeated injections of drugs. This obviously is a grave type of complication, but it can quite often be successfully managed throughout the patient's remaining lifetime.

F. Superior Vena Caval Obstruction

The superior vena caval (SVC) syndrome today is almost always due to underlying malignancy. In the past, tuberculosis and other granulomatous diseases were common causes for the syndrome. Bronchogenic carcinoma, non-Hodgkin's lymphomas, Hodgkin's disease, and primary mediastinal tumors are the most common causes now for this syndrome. The clinical features include fullness of the face, periorbital edema, plethora, headache, nosebleed, and distended neck or arm veins. The syndrome may develop rapidly or insidiously and can potentially result in massive cerebral hemorrhage. The diagnosis is usually not difficult to establish on clinical grounds, but emergency radionuclide scans or bilateral brachial venography will confirm the diagnosis, localize the area of obstruction and aid in the planning of treatment. Emergency treatment with radiation therapy or chemotherapy will often bring symptoms under rapid control. Occasionally, it is necessary to institute treatment without a specific tissue diagnosis until the SVC syndrome subsides. Thereafter, more specific treatment can be based upon the exact type of tumor. Rarely, mediastinal tumors may cause inferior vena cava obstruction.

G. Pericardial Tamponade

This is a rare complication of cancer but is occasionally seen in patients with lymphoma, breast cancer, lung cancer, or other primary mediastinal tumors. We have seen three cases of acute pericardial tamponade with malignant effusions in the last four years at the Arizona Medical Center. The causes in our three cases were: 1) breast cancer, 2) mesothelioma, and 3) a primary germ cell tumor of the mediastinum. Occasionally, acute radiation pericarditis (e.g., after mantle radiotherapy for Hodgkin's disease) may also result in cardiac tamponade. The usual clinical findings of cardiac tamponade are often present: reduced pulse pressure, elevated venous pressure, pulsus paradoxus, Kussmaul's sign, a quiet precordium, an enlarged cardiomediastinal silhouette, and occasionally, a pericardial friction rub. Echocardiography is quite useful in establishing the diagnosis. Diuretics or phlebotomy are contraindicated and the treatment of choice is emergency pericardiocentesis. This is preferably performed under controlled conditions such as in a cardiac catheterization laboratory. Installation of thioTEPA or nitrogen mustard into the pericardial space if malignant cells are present in the fluid may often prevent recurrence of the fluid (Figures 1 and 2). In some cases, a pericardial window or pericardial stripping will be necessary for long term palliation.

III. Emergencies Due to Treatment of Tumor

Most of these conditions are the result of antineoplastic treatment: chemotherapy, radiation therapy, or both. However, on occasion they can be due to extensive marrow replacement by tumor, as in leukemia, multiple myeloma, or lymphoma.

A. Neutropenia

The risk of serious bacterial infections goes up considerably when the absolute granulocyte count is less than 100 granulocytes per mm^3 of peripheral blood. The risk of infection is also proportional to the length of time during which the patient remains neutropenic. For example, it is not uncommon for the absolute granulocyte count to be low for two or three days after adriamycin-cyclophosphamide therapy for breast cancer, but this seldom results in clinical infection.[3] On the other hand, patients with acute leukemia whose bone marrows are made aplastic by the use of anti-leukemic treatment and who remain severely granulocytopenic for three to four weeks during their treatment virtually all become infected. In general, the patient who has granulocytopenia with fever should be considered infected. The usual organisms are gram negative organisms; Pseudomonas is particularly virulent. Death from sepsis can occur before the blood cultures are reported positive by the laboratory. Accordingly, our own approach to patients with granulocytopenia who develop fever is to initiate an immediate and thorough search for signs or sites of infection, obtain cultures from all possible sites, and initiate broad-spectrum antibiotic coverage. We prefer a combination of a cephalosporin and an aminoglycoside. Carbenicillin, which is particularly effective against Pseudomonas, may also be included in this combination. Even though no source of fever is found, we customarily continue antibiotic coverage for at least 7 days. If the patient remains febrile on antibiotics for longer than 72 hours, it has also been our practice to support the patient with granulocyte transfusions.[5]

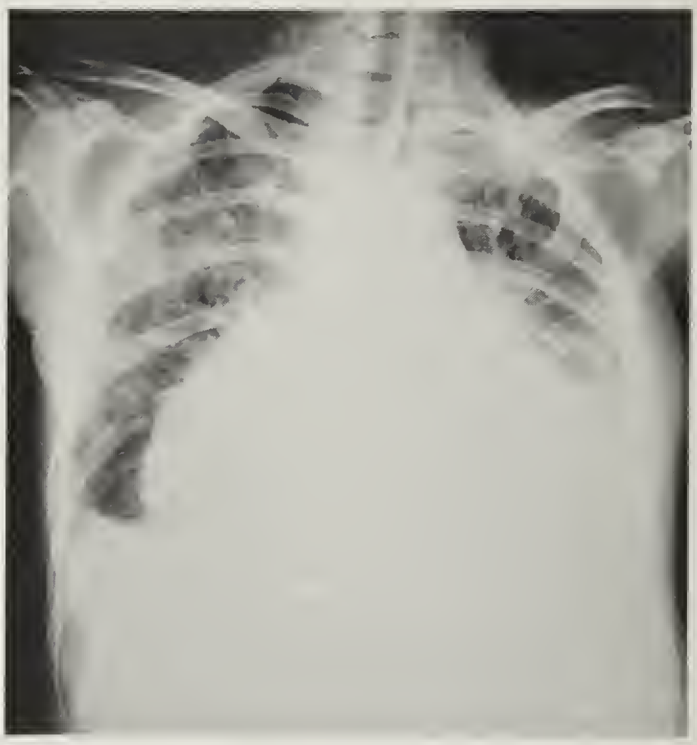

Figure 1

Chest radiograph from a 46-year-old woman with metastatic breast cancer who presented with acute pericardial tamponade.

B. Thrombocytopenia

Severe thrombocytopenia (platelet count of less than 10,000 per mm^3) is a life-threatening emergency. These patients can develop spontaneous central nervous system bleeding which may be fatal, but which is probably preventable through the use of prophylactic platelet transfusions. Our own policy on these patients is to transfuse thrombocytopenic patients who are actively bleeding or to prophylactically give platelet transfusions to those patients whose platelet count is less than 10,000 per mm^3 of peripheral blood.

IV. Rare Syndromes

A. Coombs' Positive Hemolytic Anemia

Rarely in association with solid tumors but more frequently in association with underlying lymphoma or chronic lymphocytic leukemia, we see patients with severe antibody-mediated (Coombs' positive) hemolytic anemias.[6] This can be a life-threatening syndrome. High-dose corticosteroids and sometimes splenectomy, are necessary to control hemolysis. Treatment of the underlying malignancy is also of critical importance in providing long-term palliation.[6]

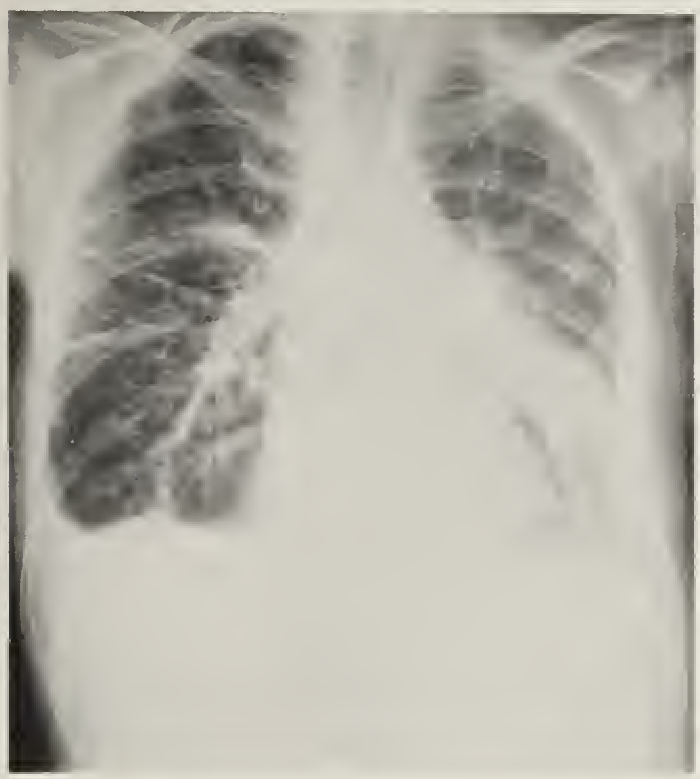

Figure 2

Chest radiograph from the same patient (on the same day) 2 hours after emergency pericardiocentesis removed 800 ml of fluid containing tumor cells. Nitrogen mustard was instilled in the pericardial sac and the pericardial effusion never recurred.

B. Immune Thrombocytopenic Purpura

The syndrome of immune-mediated excessive peripheral destruction of platelets is also occasionally seen in association with underlying lymphoma or chronic lymphocytic leukemia.[6] Treatment consists of high-dose corticosteroids, splenectomy, or both. Controlling the underlying lymphoma is obviously also important.

C. Mixed Cryoglobulinemia with Purpura

As in the conditions described above, mixed cryoglobulinemia is seen in association with underlying lymphoma or chronic lymphocytic leukemia. Circulating immune complexes are responsible for a purpuric syndrome, immune complex nephritis, or vasculitis. Treatment should include emergency plasmapheresis to remove the circulating immune complexes, high-dose corticosteroids, and splenectomy.

D. Macroglobulinemia

Occasionally monoclonal M-components of the IgM class (macroglobulinemia of Waldenström) result in a hyperviscosity syndrome with sludging of red cells, thrombosis, and potentially fatal vascular complications. Treatment is similar to that for mixed cryoglobulinemia. The underlying disease in this case is a B-cell neoplasm not unlike chronic lymphocytic leukemia or multiple myeloma. Cytotoxix therapy of the underlying condition will generally result in long-term control of the hyperviscosity once the emergency measures have reduced serum viscosity.

E. Severe Polycythemia

At high red cell masses, red cell hyperviscosity and sludging of the blood occurs which can lead to arterial thrombosis. This is a medical emergency which requires immediate phlebotomy. Thereafter, a thorough search for the cause of the underlying polycythemia should be made.

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Obstetrics and Gynecology

THE “CESAREAN BIRTH” GROUP

MARY HOUDEN, RN, MS

DONALD J. ZIEHM, M.D., EDITOR

Consumer education and participation, like it or not, is a fact of life today. Its effects have been far reaching, greatly influencing the modern practice of obstetrics. Many obstetricians consider this an impediment to their practice. New and different problems (mostly non-medical) have been imposed upon the practitioner. The prepared childbirth classes have “informed” many couples about the labor and delivery process with varying degrees of accuracy and from differing perspectives. The tendency is to blame the instructor or the method when a patient seems to be misinformed or holds a view different from the obstetrician concerning the conduct of the labor and delivery. “Educated couples” present specific goals and expectations for their childbirth experience. Questions are the order of the day. For many physicians this may be the first time a patient has asked so many questions (or questioned so many answers) about what what was previously felt to be the “Doctor’s business.” “The patient is dictating obstetrical practice with minimal amounts of knowledge” —a common complaint.

But let’s examine this problem from the patient’s side of the desk. First, the desire for more education came as an expressed need from the consumer, *the patient.* “What happens to my body during pregnancy?” What can I do to make the pregnancy experience a pleasant one for me?” Instead of passively *enduring* the *pain* of labor she wants to *actively experience* and *share* the *joy* and discomfort of the childbirth experience with her husband. In the enthusiastic quest for knowledge, information is sometimes misinterpreted, and physicians are plagued with questions that seem irrelevant.

Today’s patients are making demands about having children in a certain way which can be somewhat disturbing and *seems* to challenge the authority of the physician. His authority seems to be challenged, but in most instances the patient is going to her doctor because he possesses certain knowledge and skills needed to assure a successful outcome to the pregnancy. The consumer is asking: “Please include us in the decision-making process about things that affect us and our growing family. Don’t just tell us what you are going to do (or do what you are going to do) without even informing us. It makes us feel like non-participating robots.” This approach comes as a shock to many physicians. On the bright side is the fact that the doctor today can assist and educate the patient in a way that has never been available before. She wants to be made aware of the problems and concerns, the ‘gray areas,’’ and the alternatives in dealing with her situation.

Obstetrics is a “healthy” rather than a “sickness” specialty. When all is normal, labor progresses in a relatively predictable manner. However, this is not always the case, and many things can occur to change the course of normal labor. The possibility of a Cesarean Section is difficult for many patients to handle. The emergency nature of this surgery is partly to blame for this problem. Even though the obstetrician may have spent a considerable amount of time with the patient before arriving at the decision, consider the situation: a woman who has been in labor, is tired and in pain, suddenly has to face the possibility of a major operation, and deal with the feelings associated with that surgery, i.e. fear for her life and the life of the baby. In that situation perceptions are not the sharpest and misunderstandings occur as a natural outcome of the stress and crisis situation.

It is often difficult for the obstetrician to fully comprehend the impact of a Cesarean Section upon a woman. She is healthy, the baby is healthy, so what’s the big problem?! A moment’s reflection, “walking a mile in her shoes,” may give a somewhat different outlook. The feeling of “detachment” from the baby is common in women delivered by Cesarean Section, since she was unable to participate in the delivery experience. Difficulty is often encountered in coping with the concept that “I couldn’t do it myself,” “My body let me down.” Add to these feelings the knowledge that the next delivery will also have to by Cesarean Section, and one very apprehensive patient is produced.

Reprint requests to Dr. Ziehm, 1728 W. Glendale Ave., Phoenix, AZ 85021.

To assist patients in dealing with these and other concerns about Cesarean Section a new group has been formed. This group is meeting monthly, inviting physicians and nurses to speak to them about various aspects of the Cesarean experience. Classes are available for couples who have attended prepared childbirth courses to make them aware of the indications for Cesarean birth. Additional instruction is provided for the woman who has had a previous Cesarean Section. By providing the patient with education about the procedure, and a chance to "rap" with other women who have undergone a similar experience, it is hoped that her acceptance of this method of delivery will be more complete.

All health professionals and should recognize the needs of the consumer as valid in their eyes and work cooperatively with them. By being knowledgable about, and willing to participate with, such a group as the Cesarean birth group, there is much to be learned about the needs and fears of patients requiring this method of delivery. Too often physicians and nurses are placed in a position of having to guess or assume what the patient's needs are, then work to meet those assumed needs. Most patients want to make their needs known. If allowed to happen, the patient-physician relationship has to improve.

Seminars in Endocrinology and Metabolism

SIMPLICITY LEADS TO COMPLEXITY: ADVANCES IN CLINICAL THYROID PHYSIOLOGY

MARSHALL B BLOCK, M.D., EDITOR

Continuing with the present issue of Arizona Medicine is the series of articles entitled "Seminars in Endocrinology and Metabolism." The purpose of these short review articles is twofold. First, due to the rapid proliferation of new knowledge in the field of endocrinology and the multiple tests available for their evaluation, short, clinically oriented reviews would enable the physician to keep abreast of these newer developments as they relate to their practice. In addition, with great stress being placed on voluntary recertification in many subspecialties, reviews such as they could serve as an authoritative, succinct teaching forum. The editors will endeavor to accomplish these goals by utilizing the talents of practicing physicians as guest contributors to this series. Feedback, both positive and negative, is encouraged in order to help us fulfill these objectives.

In preceding Seminars in Endocrinology and Metabolism mention has been made of the recent finding that the majority of triiodothyronine (T_3) is formed from the peripheral de-iodination of thyroxine (T_4). This is in contrast to preceding views which thought initially there was only one thyroid hormone, thyroxine. Then T_3 was discovered and both T_4 and T_3 were thought to be predominantly made in the thyroid gland and secreted together into the blood stream. This new understanding of thyroid hormone biosynthesis has led to the suggestion that thyroxine may be a pre-hormone or prohormone in the biosynthesis of the active principal, T_3. As in every biological system, initial simplicity gives way to complexity. With the discovery of the peripheral de-iodination of T_4 to T_3, came the discovery that the removal of one iodine molecule from T_4 may take place in one of two ways giving rise to two varieties of T_3, one in which iodine molecules are in the 3, 3^1, 5 positions of the phenylalanine ring and in the other on the 3, 3^1, 5^1 positions. The latter molecule has been called reverse T_3 (rT_3).[1] The enzyme systems responsible for T_3 and/or rT_3 generation must be under regulatory function. For instance if all T_4 was routed into rT_3 rather than T_3 production, the patient would become hypothyroid for it has been found that rT_3 has little biological activity. This would result in stimulation of the hypothalamus. Thyroid releasing hormone would be made with resultant increased thyroid stimulating hormone production from the pituitary gland. This in

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Reprint requests to 3411 North Fifth Avenue, Phoenix, Arizona 85013 turn would stimulate the thyroid gland to produce increased quantities of thyroxine (T_4) which in turn would be converted to rT_3. The control of the conversion of T_4 into T_3 and/or rT_3 is not known with certainty but several clinical conditions have been studied which shed some light on this most important process(es).

For instance, during the administration of glucocorticoids there is a decline in serum T_3 levels with a corresponding increase in rT_3.[2] Likewise, in the normal fetus and newborn, several kinds of systemic illnesses of a chronic nature and complete starvation, there appears to be an increased production of rT_3 with a decline in T_3. This inverse relationship between rT_3 and T_3 may have biological significance for, during states of stress, when metabolic rates may be increased from other causes, the effect of thyroid hormone is blunted. Likewise, in states where conservation of energy is required, production of rT_3 rather than T_3 would result in decreased metabolic activity of the normally secreted thyroxine molecule. This would tend to diminish the catabolic response to such a situation, i.e., starvation. Thus, it would appear that regulation of thyroid hormone blood levels involves not only the hypothalamus and pituitary gland but also the peripheral enzyme systems which regulate T_3 and rT_3 production.

An additional advance in the understanding of the etiology of hyperthyroidism has been the finding that the pituitary gland is not alone in the production of thyroid stimulating hormone. As early as 1955 investigators first published the possibility that a compound having similar qualities was made in the placenta. In 1960 researchers noted the relationship between the development of hyperthyroidism and the occurrence of a placental hydatiform mole. In 1969 Hershman and Stahns extracted and characterized mole thyrotrophin material. They later showed that it was slightly different from pituitary TSH, but that it could stimulate thyroid gland production of thyroxine. Ultimately in the 1970's Hershman and his colleagues further described two pregnant women with severe thyrotoxicosis secondary to hydatiform mole production of placental TSH. Removal of the latter tumor resulted in regression of the hyperthyroid state. These observations have been duly confirmed by others leading to an editorial in the *Journal of the American Medical Association.*[3] The editorial makes several important observations which have a great deal of clinical relevance for physicians caring for pregnant women. The editorial asks, "how many pregnant women have born the trial of a toxic thyroid without the physician's knowing its cause? How much iodide, methimazole, and radioactive iodine have been wasted? How many thyroidectomies?" Obviously pregnant patients who have diffusely enlarged thyroid glands and are thyrotoxic by the usual peripheral blood parameters should have excluded first the presence of a hydatiform mole. This is essential if appropriate therapy is to be instituted for if the thyroid gland itself is treated and the cause for the excessive thyroid hormone secretion not investigated patients may be mismanaged. Obviously removal of the hydatiform mole will under most circumstances cause regression in the hyperthyroid state. Obviously one must make certain that Graves' disease and a hydatiform mole are not coexistant but this will be readily apparent once the mole is removed for the hyperthyroid state will not remit under those circumstances and appropriate therapy can then be instituted for Graves' disease. If uncertainty exists as to the presence of a hydatiform mole early in pregnancy then conservative measures to cause regression of thyroid hormone secretion would be in order. Thus, the use of various antithyroid drugs could be instituted early rather than the use of more aggressive maneuvers such as surgery and/or the use of radioactive iodine. Again, initial simplicity leads to complexity.

As a reminder to an observation made in these columns last year,[4] appropriate replacement doses of thyroid hormone under usual circumstances should not exceed 0.2 mg. a day of thyroxine (T_4). The use of thyroid extracts, various forms of thyroid hormone linked to thyroglobulin (Proloid) and synthetic combinations of T_4 and T_3 (Euthroid, etc.) have been shown to produce higher than normal levels of T_4 and T_3 and thus are not physiological. In addition, the use of thyroid hormone in weight reducing programs should be discouraged as a hyperthyroid state is needed to produce weight loss and this is biologically dangerous. The continuing occurrence of excessive iatrogenic thyroid hormone administration to obese patients is highlighted by a recent patient who was admitted to a local hospital severely thyrotoxic, in marked congestive heart failure with rapid atrial fibrillation due to the ingestion of almost 12 0.15 mg. tablets of T_4. Similarly, the use of thyroid hormone empirically for patients who have oligomenorrhea or menometrorrhagia should be discouraged. A recent report has appeared which has linked the ingestion of thyroid extract with the occurrence of breast carcinoma. Whether this study is meaningful only time will tell, but it and others like it, which have appeared with increasing frequency lately, emphasize that medication should only be prescribed when it is truly indicated. Furthermore, the dosage should be the smallest necessary to produce the desired effect.

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Seminars in Chest Medicine

OXYGEN TOXICITY AND BRONCHOPULMONARY DYSPLASIA

H. KENNETH FISHER, M.D.

This continuing series of articles entitled "Seminars in Chest medicine" will attempt to keep the reader abreast of developments in the broad field of pulmonary diseases. The format used will be that of brief succinct reviews written by the editors as well as guest contributors. Areas of controversy as well as practical chest medicine will be explored. We hope that these reviews will be of value in promoting continuing education for certification examinations as well as a forum for new and controversial issues. The editors welcome comments and discussion from our readers.

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To place in perspective our body needs for oxygen, we shall review briefly our major expenditures of energy. First is the need to maintain an orderly body structure in a world in which everything tends toward disorder—entropy tends to increase. Second, we need to perform external work on our environment—such as keeping our body erect during lectures even when the lights go out—and performing the walking, lifting, carrying and other physical activities of our daily lives. Finally, we need to perform a great deal of chemical work, for example, to keep potassium ions inside cells and sodium ions outside cells. To accomplish these many energy-requiring tasks, we must generate enormous numbers of adenosine triphosphate (ATP) molecules every minute, and these come principally from the oxidation of our foodstuffs.

In order to oxidize foodstuffs efficiently we must have constant access to molecular oxygen. For example, if we metabolize glucose anaerobically to lactate we recover 52 kilocalories per mole of glucose, but we recover 686 kilocalories per mole of glucose if we are able to oxidize it completely to carbon dioxide and water. In order to oxidize our fuels in a controlled manner (and thus avoid lighting up like an incandescent lamp!) we have developed the mitrochondrial cytochrome apparatus. As our foodstuffs are metabolized through the Krebs cycle, we form reduced pyridine nucleotides (NADH and NADPH, formerly known as DPNH and TPNH). As these reduced nucleotides are cycled by reoxidizing, their electrons are transferred in a highly organized way from one cytochrome to another until the electrons are passed finally to molecular oxygen, which serves as the ultimate electron acceptor. This electron transfer takes place in the cristae of mitochondria found in nearly all our tissues. Two or three ATP molecules are formed per electron pair thus transferred. It is at the mitochondrial site for final electron transfer to oxygen that we must deliver adequate supplies of oxygen.

As Robin reminded us in 1966[1] there is a large difference in oxygen tension in the air we breathe compared with that inside cells, and the oxygen tension at the mitochondrion is even lower than that in the cells in general. For example, the inspired air contains oxygen at a partial pressure approximately 130 mm mercury at Tucson (elevation 1500 feet), but intracellular oxygen tensions are around 15 mm mercury, and mitochondrial oxygen tensions are roughly 5 mm

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mercury. Fortunately, this enormous gradient does not normally constitute a problem because the speed of electron transfer from the mitochondria (from the enzyme cytochrome oxidase) to oxygen is maximal until the O_2 tension (pO_2) falls below 3 mm mercury.[2] Oxygen continues to accept electrons from mitochondrial cytochromes at slower rates until pO_2 falls below 1 mm mercury.

The problem faced by clinicians aiming to maintain even these very low oxygen tensions at the mitochondrial level is the occasional need to expose the lungs to much higher concentrations of oxygen. This difficulty becomes exaggerated when our patients have an effective right-to-left shunt of blood flow through the lungs. A patient with an effective 20% right-to-left shunt (for example one with lobar collapse or pneumonia) would thus have an arterial O_2 tension of about 60 mm mercury breathing room air and might be able to increase his arterial oxygen tension by about 100 mm mercury if he were breathing 100% oxygen. If the shunt fraction is 30% of the pulmonary blood flow, then the arterial oxygen tension (when he breathes room air) will be approximately 50 mm mercury, and will increase to only 75 mm mercury while breathing 100% oxygen. Under such circumstances oxygen injury to the lungs may occur even though blood (and mitochondrial) levels are well above normal.[3,4]

We will now consider the harmful effects of oxygen on the lungs. Generally, as inspired oxygen tension increases these abnormalities develop at faster rates. The specific lesions of oxygen poisoning vary from one organ to anothr and from one tissue type to another. They are different in the adult compared with the neonate. They are different for subatmospheric concentrations of oxygen compared with hyperbaric oxygen. They are altered by a number of drugs[5] and they are affected by certain diseases which may be present during the period of hyperoxia. For this discussion we will focus on oxygen tensions below one atmosphere, since this is the range of interest in most clinical practices. I will cite data derived from man wherever possible, and will provide supplemental information derived from animals where necessary.

What are the functional changes resulting from hyperoxia in man? Within ten hours normal volunteers breathing nearly pure oxygen at one atmosphere pressure develop retrosternal tickling sensations, noticed especially during deep breathing. They develop an occasional cough.[6] With additional hours of continuous exposure they show evidence of increasing carinal irritation and more frequent cough. The symptoms may progress by 24 to 36 hours to dyspnea at rest, and after 48 hours of exposure they progress from restlessness to lethargy, and may include anorexia and vomiting.[7] The longest survival reported in man breathing 100% oxygen at one atmosphere is approximately four and one-half days.

The earliest functional change so far described in normal man is a decrease in tracheal mucus velocity, seen within three hours of continuous exposure to 90% oxygen at sea level.[8] Caldwell and co-workers[9] studied healthy volunteers for a period of several days in an oxygen exposure chamber. Vital capacity decreased during the first day of continuous exposure to 98 percent oxygen and recovery of vital capacity to control values often required weeks following a period of several days of hyperoxia. Other abnormalities of lung function included a fall in functional residual capacity, lung compliance, and inspiratory flow rates. In contrast, airway resistance, expiratory flow rates and the alveolar-arterial gradient for oxygen were not abnormal.

There is strong evidence that resistance to infection is decreased by hyeroxia. In 1970 Huber and co-workers[10] found that mice exposed to one atmosphere of oxygen rapidly lost the ability to kill inhaled staphylococci. It has subsequently been shown that the pulmonary alveolar macrophages are still capable of ingesting the bacteria, but not of killing them. More recently, Fenters[11] has shown that animals recover quickly from this aspect of hyperoxic lung injury: 24 hours of air breathing after a single day of breathing 100% oxygen is sufficient for recovery of normal intrapulmonary killing of bacterial aerosols. In contrast, defenses against viral challenge are more seriously impaired. For example, animals challenged with an aerosol of influenza A/pr/8 virus showed increased morbidity and mortality during 60% O_2 breathing even when the hyperoxia started two to three days after the viral challenge.

Another functional effect of hyperoxia was demonstrated by Burri and Weibel,[12] who exposed newborn rats to 40% oxygen for three weeks. These rats failed to achieve normal total lung capacity as adults, even though their lungs were histologically normal. It appears from these studies that hyperoxia during development of the lung can lead to decreased lung growth.

So far our discussion has centered mainly on oxygen levels near one atmosphere pressure at sea level. It seems clear that for many adverse effects longer exposure to lower oxygen tension yields the same abnormal results. What is not clear is whether there is any truly "safe" level of hyperoxia. There is considerable disagreement on this point, and perhaps the different answers given by different investigators depend on which index of hyperoxic lung damage they have examined.

Anatomic evidence of oxygen injuries is manifest throughout the structure of the lung. Ordinarily the small bronchi are covered more or less completely with a mucus blanket beneath which lie ciliated columnar epithelial cells and mucus cells. As we have noted, the earliest change yet described during hyperoxia is the malfunction of tracheal mucus flow—although it is not yet clear whether this represents abnormal ciliary movement or abnormal mucus characteristics. Experimental reports suggest increased quantities of bronchial secretions during exposure to hyperoxia.[13] In later stages of oxygen poisoning, the mucosa of small bronchi may become necrotic and slough from the basement membranes.[13]

Beyond the respiratory bronchioles are clusters of alveoli, on whose surface lie the pulmonary alveolar macrophages whose malfunction we noted previously. Fenters' group[14] has recently described anatomic changes in the surface of alveolar macrophages, which lose their normal luxuriant fronds during prolonged exposure to hyperoxia. Whether this anatomic change has any functional significance is not yet known.

In the classic view of an alveolar septum seen in cross section, alveolar air is separated from the blood by thin cytoplasmic extensions of the type one alveolar lining cells, by the normally thin interstitial space which carries exuded fluid back into the lymphatic circulation, and by the thin endothelial cell cytoplasm. Recent investigations suggest that a more realistic view of the alveolar architecture would show the alveolar capillaries and their included red cells bulging into the air space. The degree of protrusion into the air space may have been exaggerated in some studies due to dessication of the tissue in the process of preparation, but in any case it is clear that type one alveolar lining cells and capillary epithelial cells have a very large surface area exposed intimately to the alveolar hyperoxic gas. It has been suggested that for these reasons they should manifest toxic effects of hyperoxia early as indeed has been demonstrated.

Dissecting the effects of hyperoxia on the human lung has been difficult. Several studies by Pratt[15] and Nash and co-workers[16] have described the basic observations. Patients dying with pulmonary oxygen toxicity typically have beefy, congested lungs with histologic findings that are characteristic if not pathognomonic. In adults there appear to be two phases of this illness: an exudative or "wet" phase, and a later proliferative phase. These findings have been correlated with exposure to high concentrations of inhaled oxygen, although in nearly every case there are other complicating factors which make it impossible to prove conclusively in any patient the role of oxygen in producing these changes.

Since the orderly study of the sequence of changes during pulmonary oxygen poisoning is impossible in man, we now turn to two careful and extensive animal studies of morphologic changes during continuous exposure to one atmosphere of oxygen.[17,18] After two days in an atmosphere of 100% oxygen, rats brought back to room air appeared dyspneic. Their lungs showed interestitial edema and they had pleural effusions. By three days rats were cyanotic and gasping even in oxygen, and could not survive air breathing. Histologically they had damage evident in the endothelium and type one epithelial cells.[17] Calculations of the authors suggested that by this time 65 percent of the alveolar surface had been rendered ineffective due to pulmonary edema. The calculated diffusing capacity for carbon monoxide in these animals is less than ten percent of the expected control values. In general, rats do not survive much longer than three days breathing pure oxygen, but die in this exudative or "wet" phase of pulmonary oxygen toxicity.

In contrast, monkeys have been studied by Kapanci and co-workers[18] for periods up to twelve days breathing 98 percent oxygen. In these animals the histologic changes in the capillary endothelium are limited to focal swelling. By four days type one alveolar lining cells show severe injury, and about 90% are destroyed. They separate from the basement membrane and at these sites fibrin begins to form. Since fibrin is a major constituent of hyaline membranes[19] the presence of fibrin deposits at the alveolar surface in this connection is of interest. The widened interstitial space in the alveolar septum by four days included not only edema fluid but a modest increase in collagen fibrils laid down by active fibrocytes.[5] In contrast to the type one epithelial lining cells the type two epithelial lining cells appear bigger at this time. By seven days the type one cells have been replaced by type two alveolar lining cells. The epithelium is now three times thicker than normal and the interstitial space has nearly doubled in thickness. In the interstitial spaces in addition to edema are hemorrhage and active fibroblasts with collagen fibrils. By twelve days of continuous oxygen exposure all alveolar surfaces are covered by type two lining cells, which thus increase their total volume by more than 20-fold. The epithelial barrier is now seven times as thick as normal. Interstitial pulmonary edema has yielded to extensive lung fibrosis.

Sequential studies of rats and monkeys exposed continuously to nearly pure oxygen have thus shown damage to endothelium and type one epithelial lining cells. Interstitial edema gives way to cellular proliferation and fibrosis but the animals typically die before this process goes on very long. In contrast monkeys survive the "wet lung" phase; their type two alveolar lining cells proliferate and recover the denuded alveolar surfaces. Since they survive the stage of pulmonary edema, monkeys live long enough to develop progressive interstitial pulmonary fibrosis. The changes noted retrospectively in human autopsy series appear thus to resemble those observed by Kapanci and co-workers in the monkey.

The time course of lung tissue changes during hyperoxia can be altered by prior sub-lethal exposures to hyperoxia. This phenomenon has been called "adaptation" although it may not represent an entirely benign response to hyperoxic stress.[20] Among the drugs which seem to protect lung tissue from hyperoxia are vitamin E and adrenergic blocking agents.[7] In addition to viral and bacterial infections, other agents which cause hyperoxic stress to have increased morbidity and mortality include Paraquat,[21] catecholamines and adrenocortical hormones.[7]

The term bronchopulmonary dysplasia was used in 1967 by Northway, Rosan and Porter[22] to describe a syndrome in newborn infants exposed to more than 80 percent oxygen for 24 hours or longer. With treatment lasting more than 150 hours in the 80 to 100 percent O_2 range most infants developed chronic or fatal pulmonary injury. The authors described four phases or this syndrome.

I. In the first three days of illness it looks clinically like other forms of acute respiratory distress syndromes. There is mucosal necrosis, hyaline membrane formation, atelectasis, and dilated pulmonary lymphatics. Chest roentgenograms show a fine grained appearance with air bronchograms usually visible. Cytologic examination of material recovered during routine clinical aspiration of the trachea and airways shows well preserved columnar epithelial cells with cilia in place.

II. Four to ten days after onset of illness, continuing necrosis with loss of epithelial cilia is seen. Lymphocytes become prominent in the airways, suggesting the onset of repair phenomena, Chest roentgenogram is more radiodense and exfoliated cells now are without cilia.

III. During phase III (ten to twenty days of illness) there is well developed ulcerative bronchiolitis with sloughing of airway mucosa, increased secretion of mucus, and the appearance on chest roentgenogram of irregular loose and dense areas in the lungs. Exfoliative cytology now reveals sheets of cells with vacuolated cytoplasm and prominent mitoses.

IV. Following 150 hours of exposure to nearly pure oxygen or following much longer exposures to lesser concentrations of O^2, infants typically manifest rales, cyanosis in air and often have severe cor pulmonale. The bronchial lining often appears dysplastic with abnormal nuclei and cellular size. There is interstitial fibrosis and distended respiratory bronchioles. Pulmonary arteries and bronchial arteries may be thickened. The appearance resembles that of centrilobular emphysema. Chest roentgenogram often appears hyperaerated, and exfoliative cytology shows bizarre cells initially, with a gradual return toward normal morphology.

It appears likely that early diagnosis of impending bronchopulmonary dysplasia can be established by use of exfoliative cytology as suggested by D'Ablang and co-workers,[23] but what therapeutic steps should be taken in the face of such a presumed diagnosis are not yet clear.

SUMMARY

Oxygen can injure all parts of the lung structure. In man two phases of injury are recognized: a "wet" or exudative phase and a later proliferative phase. Even within a few hours, healthy men breathing pure oxygen at sea level show functional abnormalities of tracheal mucus flow. In the newborn lung prolonged exposure to hyperoxia may have still other harmful effects. Like all drugs, oxygen would appear to be best used by whatever methods accomplish the therapeutic objective with the least exposure to hyperoxia.

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Renal Disease

ACUTE RENAL FAILURE - II MANAGEMENT OF ACUTE TUBULAR NECROSIS

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This review on management is primarily concerned with acute tubular necrosis (vasomotor nephropathy). However the principles in management also apply to acute renal failure of other types, eg., renal cortical necrosis, renal vasculitis, etc. The care of the patient with acute vasomotor nephropathy involves treatment of (1) associated or precipitating circumstances leading to renal failure such as surgery or trauma and (2) the metabolic consequences of renal failure itself. Proper care of this group of patients often requires a multi-disciplinary approach.

The metabolic abnormalities of acute renal failure are well recognized. A proper fluid balance is essential to avoid extremes of overhydration and pulmonary edema and dehydration and hypovolemia on the other hand. Replacement of insensible losses of 400 to 600 cc's per day plus replacement of urinary, gastrointestinal, and drainage losses is necessary. The measurement of daily weight, observation of tissue turgor and neck vein filling and examination for pulmonary or peripheral edema are useful signs in evaluating fluid needs. The measurement of central venous pressure in the more seriously ill patient is also of value.

Proper nutrition is important in care. It is estimated that the metabolism of acute renal failure produces a loss of body weight of between .5 to 1 pound per day. There is however a wide variation in weight loss from patient to patient depending upon the presence of fever, sepsis, caloric intake, etc. 100 grams of carbohydrate per day are recommended for maximum protein sparing. The provision of water-soluble vitamin supplements, especially in the patient receiving

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dialysis or prolonged parenteral feeding is necessary. Of interest is the recent introduction of parenteral hyperalimentation using essential amino acids and hypertonic glucose in acute renal failure. Abel, et al has reported improved survival rates and more rapid recovery from acute renal failure with parenteral hyperalimentation. This form of feeding would seem to have its greatest value in the post-surgical, post-traumatic or otherwise critically ill patient unable to take adequate oral caloric intake or who is extremely catabolic. The alternative use of hypertonic glucose with serum albumin has also been used for purposes of providing adequate nutrition. Hyperglycemia is extremely common with hyperalimentation solutions frequently requiring insulin in these patients and probably related to the acute glucose intolerance or pseudo-diabetes seen commonly with renal failure. Where possible introduction of an oral diet with modest protein restriction and sodium and potassium restriction as indicated by urinary output and serum electrolytes should be a therapeutic goal. This is often possible with the less seriously ill patient undergoing periodic acute dialysis.

Serum electrolyte abnormalities are extremely common. Hyponatremia of modest extent is common and usually requires no treatment. Severe hyponatremia with a serum sodium between 110 to 115 mEq per liter or less is usually associated with over-hydration and the use of glucose-water solutions intravenously. The attendant mortality in patients with acute hyponatremia of this severity is 50% and requires prompt therapeutic intervention with the use of hypertonic saline solution and dialysis. Primary manifestations are neurological with coma and generalized seizure activity. Hyperkalemia is another frequently encountered problem. Serial electrocardiographic changes include peaked T-waves, increased PR interval, widening of the QRS complex, idioventricular rhythm and ultimately cardiac arrest. It is frequently difficult to correlate cardiographic changes with a serum potassium alone since co-existent hyponatremia and alterations in serum calcium level will modify electrocardiographic activity as well. However the maintenance of a serum potassium of 6 mEq per liter or less should be attempted. The treatment of life threatening hyperkalemia with advanced cardiographic abnormalities include intravenous calcium gluconate, intravenous sodium bicarbonate, intravenous glucose with regular insulin, Kayexelate either orally or by retention enema and finally dialysis. Hypermegnesemia is common but is almost never a clinical problem; however use of intravenous or intramuscular magnesium, magensium antacids by mouth shoud be avoided since serious neurologic and cardiac sequelae can occur with extreme elevations of serum magnesium. Hypocalcemia is commonly seen but again is rarely symptomatic; with rapid correction of severe metabolic acidosis however uncorrected co-existing hypocalcemia can produce tetany and fatal cardiac arrest and must be corrected.

Neurologic sequelae of acute renal failure are relatively common. Asterixis, obtunding of the sensorium and seizure activity are usually due to the uremic state per se. Flaccid paralysis may occur with severe hypo- or hyperkalemia. Profound hyponatremia of less than 115 mEq per liter may be attended by coma and seizure activity. Finally neurologic symptoms may be seen with inappropriately high doses of penicillin G with obtundation and seizure activity as well. The correlation between uremic neurotoxicity and the level of BUN and or serum creatinine is a very inexact one but is usually mild or absent with a BUN below 100 mg.%.

Dose modification of many drugs is necessary in acute renal failure. Major dose reduction of aminoglycoside antibiotics, penicillin G, carbenicillin and the cephalosporin group is necessary to avoid potential toxic effects of these agents. In addition maintenance therapy with digoxin or digitoxin must be substantially reduced to avoid digitalis toxicity. However other agents such as lidocaine, either in bolus or maintenance infusion, require essentially no dose modification.

Sepsis remains one of the major causes of death in acute renal failure. Appropriate treatment and prevention of infection is important in reducing morbidity and mortality. Indwelling Foley catheters should be removed where possible. The use of indwelling intravenous catheters for feeding should be handled with care and removed promptly when oral feeding is possible. Probably the commonest source of overwhelming sepsis remains the lung complicated by the frequent appearance of pulmonary edema and the frequent need for endotracheal or tracheostomy tubes in the patient with severe respiratory compromise. Additional sources of infection and sepsis include arterio-venous cannula sites, abdominal and pelvic wounds and extensively devitalized tissue in the traumatized patient. It is of interest that Kleinknecht was unable to reduce the mortality from sepsis in his group of vigorously dialyzed patients; in contrast, mortality in acute tubular necrosis patients with gastrointestinal hemorrhage was reduced from 40% to 19%.

A second major cause of death in acute tubular necrosis and one which has probably not received adequate attention is gastrointestinal hemorrhage. Kleinknecht observed that gastrointestinal hemorrhage was either the primary or contributing cause to death in approximately 25% of those patients who died with acute renal failure and acute tubular necrosis. Of those patients who developed gastrointestinal hemorrhage the mortality rate was 40%. In those patients receiving intensive dialysis to maintain a BUN of less than 100 mg.% and in whom improved caloric and protein intake was provided, the mortality rate from gastrointestinal hemorrhage was reduced to 19%. The most common cause of gastrointestinal hemorrhage was stress ulceration. Despite surgical intervention with either vagotomy and pyloroplasty or partial gastrectomy, the prognosis remained poor with recurrence or persistence of the hemorrhage from the ulcer and often the appearance of new ulcers during re-operation or at autopsy. Brown and Cameron in their series of patients with acute tubular necrosis following surgery noted gastrointestinal hemorrhage in twelve of their seventy three patients but in only four patients was it the major cause of death. In contrast to the experience of Kleinknecht, they were unable to reduce mortality from gastrointestinal hemorrhage despite intensive dialysis on either a daily or alternate day basis.

The use of peritoneal or hemo-dialysis in treatment of acute tubular necrosis is well recognized. Despite the use of dialysis, mortality in acute tubular necrosis remains approximately 50 to 60% in reported series in the postoperative patient, approximately 65% in post-traumatic military personnel and approximately 30 to 40% in acute tubular necrosis due to obstetric and medical complications. The reduction in mortality rates reported by Kleinknecht and Eliahou with intensive hemo-dialysis have not been generally duplicated by other groups. Despite these sobering figures, the role of dialysis is quite clear in preventing and correcting a number of complications. The indications for instituting dialysis treatment include severe pulmonary edema, severe metabolic acidosis, hyperkalemia which is unresponsive to non-dialytic measures and metabolic encephalopathy. Profound hyponatremia may require the use of hypertonic saline but this measure carries a real hazard of sodium overloading and precipitation of acute pulmonary edema so that dialysis is frequently required.

The choice of using peritoneal versus hemodialysis can be individualized. While there are few if any absolute contraindications to peritoneal dialysis, it is considerably less efficient than hemo-dialysis. Potential problems include excessive protein loss via peritoneal drainage, ventilatory compromise due to basilar atelectasis and less commonly, the development of a bacterial peritonitis. Peritoneal dialysis does offer advantages in the patient who has active gastrointestinal hemorrhage in whom heparin is hazardous and in the patient who is hemodynamically unstable with cardiac arrhythmias and hypotension. Hemo-dialysis on the other hand permits more rapid correction of metabolic abnormalities as well as more rapid removal of edema fluid. While the question of adequacy of dialysis in acute tubular necrosis is not completely resolved, it seems advisable to dialyze the patient in order to maintain a serum creatinine below 10 mg.% and a BUN below 100 mg.%. It may be necessary to dialyze daily or on an alternate day basis to achieve this goal particularly in the extremely catabolic patient.

As mentioned earlier the oliguric phase of acute tubular necrosis (vasomotor nephropathy) usually lasts about two weeks although oliguria may last six to eight weeks. The oliguric phase is followed by a diuretic phase with generally stepwise increment in urinary output. Diuresis of greater than 3 to 4,000 cc's per day mentioned in older reviews is relatively unusual in our experience and tends to be short lived. The diuretic phase is attended by increased losses of sodium and potassium and these solutes as well as water losses must be replaced to prevent hypovolemia. In addition, hypercalcemia may occur during the diuretic phase and is thought to be due to secondary hyperparathyroidism but is usually transient. Long term followup of renal function following recovery from acute tubular necrosis shows that clinical recovery occurs in virtually all patients. Formal measurements of maximum concentrating ability, glomerularfiltration rates, and renal blood flow indicate that a substantial percentage of patients do not experience complete functional recovery. This is particularly true in the older age groups. This permanent loss of renal function however is rarely of clinical importance.

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AM Grand Rounds

RECENT ADVANCES IN ULCER THERAPY: SELECTIVE PARIETAL CELL VAGOTOMY

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CASE REPORT

The patient today was presented by Dr. Stanley Crie (medical resident).

A 55 year old female alcoholic was admitted to the surgery service because of the sudden development of stupor following an episode of hematemesis. In addition to several years of heavy ethanol intake the patient had an episode of hematemesis nine years prior to admission which was attributed to peptic ulcer disease with no known recurrence. On initial examination the patient was stuporous and jaundiced with hepatomegally and ascites. The gastric aspirate contained blood. Her hematocrit was 15%, hemoglobin 5.2, a prothrombin time 3.6 sec. over control, and her liver function tests were abnormal. The initial assessment was that the patient was bleeding from esophageal varices and had stage three hepatic encephalopathy.

Following transfer to the medicine service on the third hospital day the patient had upper gastrointestinal endoscopy which demonstrated a bleeding duodenal ulcer and non bleeding varices. In spite of transfusions and intensive antacid and anticholenergic therapy with intermittent gastric aspiration, the patient continued to bleed and her urinary output fell to below 500 cc's per day. Since the patient had failed to respond to a maximal medical regimen and was not a reasonable surgical risk, it was elected to institute intravenous cimetadine therapy (kindly provided by Smith-Kline and French, Inc.), on the seventh hospital day. The patient's intragastric pH fell to 7, 90 minutes after starting the drug and remained there throughout the treatment period. Three days later the patient's stools became quaiac negative. Re-endoscopy on the eighteenth hospital day revealed that the ulcer had healed. Because the ulcer had healed and concern over giving a new drug in the face of persistent oliguria, cimetadine was stopped. In spite of no recurrence of bleeding, the patient had continuing deterioration of hepatic and renal function and died on the 25th hospital day.

Dr. Mueller:

This case illustrates the possible benefit of cimetadine, a new inhibitor of acid secretion, in a difficult therapeutic situation. The ability of this drug to virtually shut off gastric acid secretion represents a major therapeutic advance and the treatment of bleeding ulcer patients who are not

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surgical candidates represents only one of its therapeutic potentials.

The search for effective therapy of ulcer disease has been guided largely by our understanding of the etiologies of ulcer disease. Evidence has accumulated from studies done at several centers that persons with duodenal ulcers secrete more acid in response to a meal[1] and they secrete more acid over night while sleeping than do persons without ulcers.[2] Duodenal ulcer patients also release more of the acid secretion stimulating hormone gastrin in response to a meal than do controls.[1]

Excessive activity of the vagus nerve and excessive release of gastrin by the antral "G cells" have been blamed for this hyper-secretion of acid. At present the cause is not known and distinction between the mechanisms is made difficult by the complex interaction between the two. Not only can vagal stimulation cause gastrin release, but vagal innervation of acid secreting parietal cells greatly enhances their response to gastrin. On the other hand, gastrin enhances the response of the parietal cells to vagal stimulation. Although our understanding of the mechanism of hyperacidity seen in duodenal ulcer patients is incomplete, our perception of hyperacidity is clear and guides our therapeutic attacks on ulcer disease.

Current ulcer therapy is aimed almost exclusively at reducing acidity, and though effective it has serious limitations. Oral antacid preparations are effective in neutralizing secreted acid, but they must be taken in large quantities and frequently as they are effective only as long as they are in the stomach. They may be significantly emptied from the fasting stomach in fifteen or twenty minutes. They, therefore, have little use in controlling night time acid secretion and require high degree of patient compliance to achieve optimal effectiveness during the day. Although it has long been held that at least antacids are effective in controlling duodenal ulcer pain, a recent comparison with placebo reveals no advantage to antacids.[3] Anticholenergic agents are effective in reducing the secretion of acid, but their usefulness is greatly limited by patient intolerance of their other anticholenergic effects such as dry mouth and impaired accommodation. Even at maximally tolerated levels they only reduce meal stimulated acid secretion by twenty-five to thirty percent.[4]

Surgical vagotomy combined with drainage procedures are extremely successful in preventing recurrence of ulcer disease with recurrence rates of five percent or below with pyloroplasty and less than two percent with antrectomy. However, one in five who have these operations will be plagued by bothersome long term side effects from their surgery.

For these reasons advances in therapy are still sought for and major improvements are exciting. I am going to discuss two major advances in ulcer therapy, one medical and one surgical. Both are aimed at reducing acid secretion and both do it effectively with lower incidence of side effects than seen with previous therapy.

H_2 Histamine Blockers

The recognition that there are two distinct histamine receptors, one the H_1 receptor, which is blocked by the classic antihistamines and a second type of receptor, the H_2 receptor, which is not,[5] opened the way for a major advance in drug therapy of ulcer disease. The H_2 receptor has actions which include stimulation of the isolated atria, inhibition of rat uterus contractions and stimulation of gastric acid secretion. Durant and his co-workers in England[6] recognized the therapeutic implication of the H_2 receptor and set about to synthesize a series of antagonists specific to the H_2 receptor. Their success not only established the existance of the H_2 receptor but provided two H_2 blochers of considerable therapeutic potential, metiamide and cimetadine.

The role of histamine in gastric physiology has largely been neglected while the roles of the vagus nerve, local cholenergic reflexes and gastrin secretion by the antral "G cell" have become established and widely explored. Code and early workers[7] elaborated circumstantial evidence that histamine normally functioned in the stimulation of gastric secretion. It was long known that substantial amounts of histamine are found the parietal cell region of the gastric mucosa, and that histamine is a potent and direct stimulant of parietal cell acid secretion. In fact, histamine has become the standard stimulant in the maximal gastric acid secretion test. More recently it has been recognized that feeding, cholenergic stimulation and gastrin administration decrease gastric mucosal histamine levels,[8] but none of these observations lead to a wide spread appreciation that histamine played a role in normal gastric acid secretion. The discovery that H_2 histamine blockers dramatically alter acid secretion in response to a variety of stimulae opened a new interest in histamine's role in acid secretion and new information will be forth coming.

Histamine is a potent stimulator of the H_1 and H_2 receptor sites while 2 methylhistamine is an H_1 stimulator with little H_2 activity and 4 methyhistamine is an H_2 stimulator with little H_1 activity.[9] Classic antihistamines (see figure 1) are compounds in which the iminazol ring is replaced by aryl rings while the H_2 blockers metiamide and cimetadine retain the iminazol ring but add a methyl group at the 4 position and alter the ethylamine side chain.

H_2 blockers are potent inhibitors of gastric secretion stimulated by all modalities yet studied. They block acid stimulation by histamine, acid stimulated by carbachol (a cholenergic) and acid stimulated by meals both in normals and duodenal ulcer patients.[10] In contrast to anticholenergics which block secretion by only 25 to 30%, H_2 blockers can inhibit 70 to 90%[11] of meal stimulated acid secretion. In addition, an H_2 blocker combined with an anticholenergic can almost completely block the marked hypersecretion of acid seen in patients with Zollinger-Ellison syndrome.[12] A single bedtime dose of an H_2 blocker also markedly reduces nocturnal acid secretion seen in normals and duodenal ulcer patients.[13] The effect of the drug in all these situations is long lasting. For instance, depression of meal stimulated acid remains at four to five hours after one dose of cimetadine.[11] These effects and their long duration of action suggest that this drug will be extremely effective in duodenal and gastric ulcer therapy when given on a three or four times a day basis. The observation that cimetadine increases the gastric mucosal potential difference,[14] a measurement shown to correlate with changes in the gastric mucosal barrier, suggests that the H_2 blockers may have effects beneficial for the healing of gastric ulcers other than their blockade of acid secretion.

A multicenter trial of the H_2 blocker metiamide in English patients with duodenal ulcer demonstrated a high rate of healing (67%) in four weeks as compared to a group receiving a placebo (25%).[15] Both groups took antacid tablets as they wished. The initial H_2 blocker tested for clinical use was metiamide. This drug was given to about two hundred patients in daily doses of up to 1500 milligrams for periods of up to six months until two cases of reversible granulocytopenia were appreciated. These were attributed to the thiourea side chain of metiamide. Cimetadine, in which the thiourea group has been replaced by cyanoguanidine has not caused granulocytopenia in animals and is now undergoing extensive trials in this country. The H_2 blockers have shown remarkably few side effects and have no associated symptoms. Mild elevations in SGOT's and SGPT's occur, but with a frequency not significantly higher than those seen in placebo treated groups.

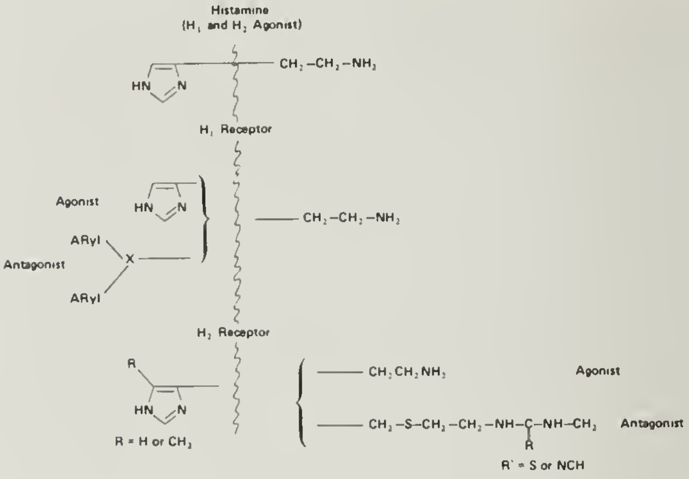

Figure 1

Relationship between Histamine and H_1 and H_2 Agonists and Antagonist (after Durant et al[6]).

In conclusion, the H_2 blockers are potent inhibitors of gastric acid secretion which have been shown to be clinically useful in the treatment of duodenal ulcer. If the change in structure that produced cimetadine leaves behind the potential to produce granulocytopenia, cimetadine promises to be major advance in ulcer therapy.

Parietal Cell Vagotomy

(Super Selective Vagotomy, Proximal Gastric Vagotomy)

Since the introduction of vagotomy in the 1940's it has become the keystone of present day ulcer surgery. When complete, vagotomy will reduce acid secretion to maximal histamine stimulation by about 70%, and will reduce the rate of ulcer recurrence to 5% or less than 2%, depending on whether or not an antrectomy is added. Further confirming the effectiveness of vagotomy is the observation that recurrences of ulcer are usually only seen when the vagotomy is incomplete. From the beginning, it was recognized that vagotomy as it was practiced was unphysiologic and that unwanted side effects could be anticipated. Truncal vagotomy severed all vagal connections to the abdomen, including those to the gastric antrum. Because an active antrum is needed for normal gastric emptying, vagal denervation leads to marked gastric stasis and a drainage procedure had to be added. Although a very high percentage of patients receiving these procedures benefited by having no further ulcers, about 25% suffered from a variety of side effects ranging from the annoying to the debilitating. Early dumping (a syndrome of symptoms of hypovolemia, abdominal cramps and diarrhea, shortly following a meal) and chronic diarrhea are most frequent. Post prandial hypoglycemia, billious vomiting, heart burn, early satiety and malabsorption are other less frequent but bothersome complications. In an attempt to overcome these deficiencies, two modifications of truncal vagotomy and gastric resection were made, the use of a pyloroplasty as a drainage procedure, and the selective rather than truncal vagotomy. Selective vagotomy spares the vagal branches to the liver and celiac plexis. Neither of these modifications resulted in a significant reduction in unwanted side effects.

A rational approach to the problem would be to sever only the vagal connections to the parietal cell containing portion of the stomach while leaving the rest of the innervation intact. F. Holle and his associates in Germany have pursued this approach from the 1960's on.[16] Others in England, Denmark and this country have used the technique, abandoning Holle's use of an added drainage procedure. Technically the procedure is said not to be difficult, but is considerably more tedious and requires more attention to detail than the conventional vagotomy.[17] Patients are said to have a very short period of post operative ileus and require no post operative gastric suction, thus reducing immediate operative morbidity.[17] Mortality rates are low, perhaps below 0.1% when done electively. Reduction in acid secretion is to the same levels as achieved with truncal vagotomy.[18] Studies of gastric emptying show that there is considerable improvement with the parietal cell vagotomy over the truncal or selective in that the pattern of gastric emptying more closely resembles normal.[19] Liquids are emptied less rapidly and solids are emptied more rapidly with the parietal cell vagotomy than with selective or truncal vagotomy. This confirms clinical observations that drainage or emptying is adequate and less than 1% have required re-operation for a drainage procedure.[17] Thus, the goal of denervation of the parietal cell while maintaining normal pyloric function appears to have been achieved.

The effects of selective vagotomy on release of intestinal and pancreatic hormones have been compared to the effects of truncal vagotomy. More pancreatic insulin is released in response to a hypertonic glucose meal and more pancreatic glucogon is released with insulin induced hypoglycemia with selective vagotomy than with truncal vagotomy,[20] confirming the impression gained in dogs that vagal innervation of the pancreas plays an important role in pancreatic islet cell function. Although there is no direct clinical correlate of these observations, it seems probable that some of the post vagotomy symptoms may be promoted by these mechanisms. A major theoretical question raised about the desirability of perserving vagal innervation of the antrum is, "What is the contribution of vagally stimulated antral release of gastrin to the hypergastrinemia seen following meals in duodenal ulcer patients?" Because antral gastrin release is very sensitive to antral pH, any measure which lowers antral pH during meal stimulated gastrin release will result in more gastrin released. Therefore, forms of successful vagotomy result in more gastrin release with meal stimulation than prior to operation. Although vagal stimulation via insulin induced hypoglycemia produces more gastrin release in patients with parietal cell vagotomy than in patients with truncal or selective vagotomy, meals produce the same gastrin release in all forms of vagotomy suggesting that vagal activity is not a particularly important contributor to meal stimulated gastrin release.[21]

Procedure	Mortality	Recurrence	Dumping	Diarrhea
Vagotomy and Resection	1.7	6%	21%	20%
Vagotomy and Drainage	0.6	1%	15%	20%
Parietal Cell Vagotomy	< 0.1	2-3%	4%	3%

Figure 2

Typical results of Vagotomy and Resection, Vagotomy and Drainage and Parietal Cell Vagotomy from published series.

Two major side effects of vagotomy and drainage procedures which are felt to be due to ablation of pyloric function, early dumping and diarrhea, are significantly less in patients with parietal vagotomy as compared with truncal or selective vagotomy (see figure 2). In studies available, bilious vomiting and nausea appear to be significantly reduced by parietal cell vagotomy.[22] These symptoms, likely due to bile regurgitation into the stomach, might be expected to be reduced by a procedure which left pyloric function intact. Other symptoms, such as hypoglycemic symptoms (late dumping), heartburn and early satiety, appear to be the same with either procedure.

Lastly and most importantly one must consider the ulcer recurrence rate with this new procedure. A major problem in this evaluation is that standard operations are judged on the basis of five year recurrency rates. The procedure has not been widely applied long enough to generate comparable five year figures. Early figures (two years) compare quite favorably with two year figures from other procedures, and it is likely that the procedure should be as effective as a vagotomy and drainage without antrectomy. One real caveat, however, stems from the documented difference in the achievability of complete vagotomy in the hands of different practitioners. These differences between practitioners appear to be real and consistent.[23] Current series of results of parietal cell vagotomies are being reported by enthusiasts who admit that the procedure is tedious. Their good results may be in part a reflection of their devotion to the procedure. We need to know if their "completeness" of vagotomy can be reproduced widely by a diverse group of practitioners. Time should tell as controlled trials are now underway. In conclusion, two direct attacks on the acid hypersecretion associated with duodenal ulcer have yielded two therapeutic modalities which are potentially major improvements. One, the parietal cell vagotomy represents the further improvement of an effective procedure by the application of phsyiologic principles. The other, the development of H_2 histamine blockers, represents not only a potentially major therapeutic advance, but also a very significant advance in our understanding of gastric physiology. Each measure offers hope to the patient with ulcer disease and to his physician. Let us hope that like so much that has gone before them they are not tarnished by time.

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Radiology Case of the Month

CASE NO. 16

TIM B. HUNTER, M.D.
KAI HABER, M.D.

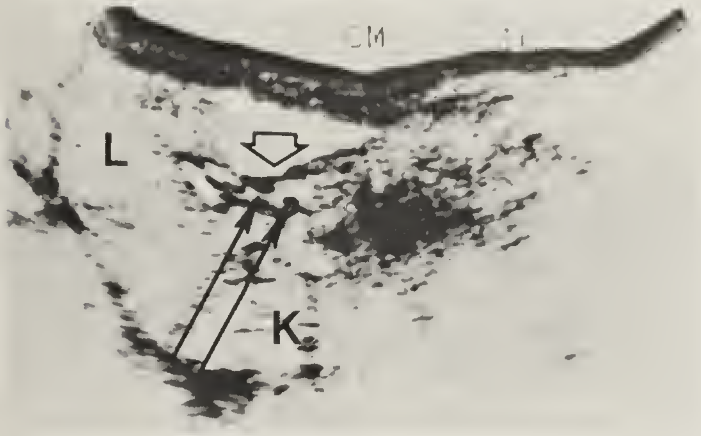

Figure 1

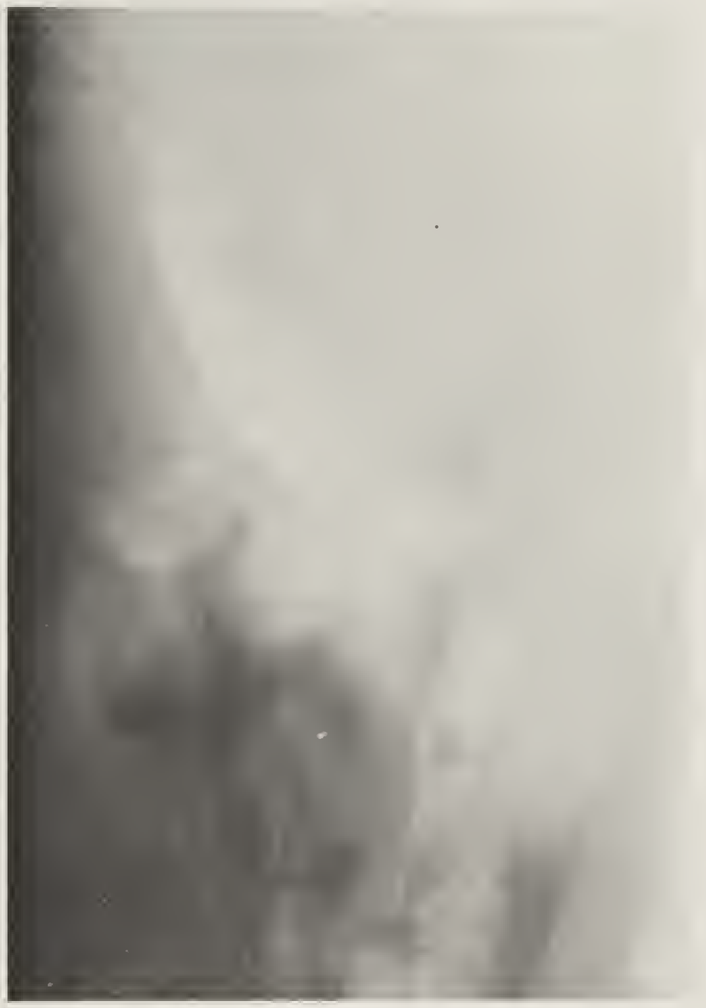

Figure 2

This case illustrates the usefulness of abdominal ultrasonography in evaluation of the gallbladder and biliary system. The patient is a 66 year old white female who presented with sudden onset of epigastric pain, nausea and vomiting. She had a past history of a bleeding gastric ulcer and was taking steroids for polymyalgia rheumatica. Examination at the time of admission revealed an elderly, well-nourished woman in moderate distress from abdominal pain. There was marked epigastric tenderness with guarding. During her initial hospitalization she was treated with nasogastric suction, intravenous fluids, and pain medication. Significant laboratory studies included a stable hematocrit of 41, markedly elevated SGOT and SGPT values, a total bilirubin of 4.3, and a moderately elevated alkaline phosphatase. Gastric aspirates and stool samples were guaic negative. An upper gastrointestinal series was also negative. Because she had had a non-visualized gallbladder on a double dose oral cholecystogram in 1975, an abdominal sonogram was performed (Figure 1). In addition, an intravenous cholangiogram (Figure 2) was attempted despite the relatively high bilirubin value. What do these studies demonstrate? How well does the ultrasound exam correlate with the cholangiogram?

COMMON BILE DUCT STONES WITH PARTIAL BILIARY OBSTRUCTION

The intravenous cholangiogram shows mild dilatation of the main hepatic and common bile ducts. Three lucent stones are visible in the common duct and the gallbladder is small and shrunken. At surgery the gallbladder was indeed chronically diseased with multiple small stones in the lumen. Four partially obstructing large stones were removed from the dilated common bile duct.

This case demonstrates the generally good correlation between the intravenous cholangiogram and surgical findings. Nevertheless, the good biliary visualization obtained on this study is unusual, since the chance for adequate visualization decreases rapidly with increasing bilirubin values. If the serum bilirubin value is above 4 mg. per 100 ml., opacification is seen in only about 9% of the cases.[1] The patient's sonogram also demonstrated the same basic pathology seen on the intravenous cholangiogram. Figure 1 represents a parasagittal sonographic section 6 cm to the right of the midline. L = liver; CM = costal margin; IC = iliac crest; K = kidney. The dilated common duct is well seen (large arrow head). Two or three echo producing areas are within the duct (long arrows). These cast acoustic shadows compatible with stones.

The excellent correlation between the cholangiogram, sonogram and surgical findings shows the usefulness of abdominal sonography in the diagnosis and management of biliary tract problems. Frequently, there is poor or no visualization of the gallbladder with orally administered contrast agents. The biliary ducts are seldom visualized with oral agents and even mild liver dysfunction can preclude biliary tract opacification by either the oral or intravenous agents. Transhepatic cholangiography and/or endoscopic retrograde cannulaization of the common bile ducts with contrast injection can provide excellent visualization of the biliary system but are time consuming procedures with some patient risk. Abdominal sonography can be performed rapidly without known harmful effects. It is a good screening examination, particularly in those patients with poor liver or gallbladder function precluding oral or intravenous contrast studies. There is disagreement in the literature as to the overall accuracy of the sonographic evaluation of the biliary system. It is probably fair to state that an experienced interpreter will have a reasonably high accuracy in those cases where definite sonographic abnormalities are seen. When the sonogram appears "normal," false negatives can be a problem. However, a recent report suggests that improved instrumentation and observer awareness of the sonographic manifestation of gallstones have made the sonographic examination highly accurate with respect to gallbladder disease.[2]

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Drug Therapy Problems

ROBERT E. PEARSON, M.S., R.Ph.

Most practitioners rely upon their own reading plus other external sources to assist them in their quest to remain abreast of the biomedical literature. This feature is intended to provide finite information, to answer some questions, and to stimulate awareness of the availability of an unbiased source of biomedical information. The format will include: questions and answers, with the questions being provided by the readers and/or users of our service; abstracts from the literature; brief descriptions of newly-marketed items; brief discussions of new innovations in therapy; and short exercises regarding specific drug products.

Q. Does cigarette smoking have an effect on theophylline?

A. Two recent studies have shown that the half-life of theophylline is significantly reduced in active cigarette smokers and may take from 3 months to 2 years to normalize after cessation of smoking. The half-life of theophylline in smokers in the two studies was 4.1 ± 1.2 hr (1) and 4.3 ± 1.4 hr (2), while that in the non-smokers used as controls was 7.2 ± 1.8 hr (1) and 7.0 ± 1.7 hr (2). The postulated mechanism is the result of induction of drug-metabolizing enzymes that do not readily normalize after cessation of smoking. Both studies were done in young subjects with the authors of one study[2] concluding that dosage levels should be about twice those needed by non-smokers.

(1) *Life Sci.* 17:195-198, 1975; (2) *Clin. Pharmacol. Ther.* 19:546-551, 1976.

Q. Any significance to a report of methadone-rifampin interaction?

A. A recent report of rifampin-induced methadone withdrawal is significant. In this report, seventy per cent (70%) of the rifampin-treated patients (21/30) in a methadone maintenance program experienced mild (14 patients) to severe (7 patients) withdrawal symptoms. Symptoms appeared 6-8 hours after combined rifampin-methadone therapy. Fifty-six (56) patients receiving anti-tubercular therapy, not including rifampin, did not experience withdrawal symptoms. While you may or may not be aware of which of your patients may be on methadone maintenance, the sensitive nature of methadone maintenance requires tact in making patients aware of this interaction.

(*NEJM* 294:1104-1106, 1976)

Q. What household products can be considered relatively non-toxic?

A. Numerous items, if consumed in small amounts, may be considered non-toxic. Some types of products are listed below:

After shave lotions*
Airplane glue+
Ballpoint pen inks
Battery (dry cell)
Bleach (Na hypochlorite
  5%)++
Body conditioners*
Bubble bath soaps/
Candles
Caps for toy pistols
  (up to one roll)
Cigarettes((
Colognes*
Crayons marked AP or CP
Contraceptive pills)
Cosmetics and skin
  preparations
Dehumidifying packets
Deodorizer cakes
Fish bowl additives
Fluoride tablets
  (<60 tablets)
Matches
  (<20 wooden or
  two books)
Model cement
Pencils
Porous tip markers
Play Doh
Putty
Roach tablets (1-2)
Sachets
Shaving creams
Silly putty
Soaps
Teething ring fluid
Thermometers#
Toilet water*
Toothpaste

*Contains high concentrations of ethyl alcohol, may be toxic in large amounts
+Not harmful unless deliberately inhaled in high concentration
++Stronger solutions (20%) found in some industrial bleaches are caustic. This concentration may produce gastrointestinal distress and vomiting

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Address all communications to the author. Mr. Pearson is Education/Research Associate with David Wastchak & Associates, Inc., Pharmaceutical Consultants, 1818 Grand Avenue, Phoenix, Arizona 85007 (602) 253-9323.

May produce vomiting or diarrhea
((Nicotine is poorly absorbed and produces vomiting
)May produce nausea and occasionally vaginal bleeding in young girls
#Metallic mercury is not harmful

(*Applied Therapeutics for Clinical Pharmacists*, Eds. L. Y. Young and M. A. Kimble, Applied Therapeutics, Inc., San Francisco, CA. 1975, p. 514)

Abstract of Interest: "Effect of Skin-Cleaning Agents on Dextrostix Readings", *Brit. Med. J.* 22 May 1976, p. 1279.

A 79 year old insulin dependent diabetic was brought to the accident and emergency department unconscious. Apart from increased muscle tone and bilateral extensor plantar responses, there were no abnormal physical signs. An initial assessment of blood glucose was made with Dextrostix reagent strips by two independent observers from samples of capillary blood obtained by finger prick. Visual readings were recorded as falling between 9.7&13.9 mmol/l (175 and 250mg/100ml). However, formal labortory estimation on a venous blood sample taken at the same time gave a value of 1.67 mmol/l (30mg/100ml) and as a consequence 50% glucose was then given intravenously, with rapid improvement in the patients clinical condition.

The Dextrostix reagent strips used were from a freshly opened bottle and when tested on venous blood were found to give accurate and reproducible results. Formal laboratory estimation of blood glucose gave substantially lower values, thus indicating that the discrepancy was in the technique used rather than in the Dextrostix reagent strips themselves. Further inquiry revealed that before the finger stab the skin had been liberally swabbed with 70% isopropyl alcohol (Sterets swab).

Ethanol or 70% isopropyl alcohol when added to blood produces a sticky brown precipitate of denatured hemoglobin that will adhere to the surface of a Dextrostix reagent strip. Possibly residual alcohol on the skin after swabbing may mix with blood to produce this brown precipitate, which can impart a brown color to the test surface. This may be interpreted by an uncritical observer as indicating a falsely high blood glucose level, particularly in poor lighting and when the true blood glucose is low. More significantly, Dextrostix reagent strips discolored by the brown precipitate also give falsely high readings when used with a reflectance meter (Eyetone, Ames Co.)"

Abstract of Interest: "Effect of Activated Charcoal on Acetaminophen Absorption", *Pediatrics* 58:432-435, 1976.

Five healthy male volunteers, 27 to 40 years old, gave their informed consent and participated in the study. Acetaminophen was administered in the morning on an empty stomach as an elixir, as a suspension, or as tablets in 1 gm doses. Activated charcoal was administered as a slurry in 200ml of water. Different dosage form experiments were carried out in random order, at least one week apart. Urine samples were collected and assayed for total acetaminophen. The availability of acetaminophen from pediatric suspension and from tablets was essentially complete, relative to that of the elixir. Ten grams of activated charcoal, given 30 minutes after 1 gm of acetaminophen decreased significantly the absorption of the drug in each dosage form: elixir, 68.9%; suspension, 49.7%; and tablet, 46.0% (relative availability to the elixir without charcoal). The same dose of charcoal given immediately after a 1 gm dose of elixir, showed a relative availability of 38.5%. This mode of therapy is recommended only as an initial step to be followed by appropriate medical assessment and treatment.

AM *Department of Health Services*

PUBLIC LAW 93-641

SUZANNE DANDOY, M.D.
DIRECTOR

The National Health Planning and Resources Development Act of 1974 (Public Law 93-641) has received more attention and comment than and recent federal legislation involving medical care.

Among the members of our own profession, the comment has been mostly critical. The criticism has its wellspring in the fear that, in a number of ways, the health planning and related laws inject a bureaucratic presence between the physician and his patient. The best antidote for criticism—the best defense against actions potentially harmful to good medical practice—is physician participation in explorations and approaches that affect the practice of medicine.

One of those approaches is Public Law 93-641. The major goal of the legislation is to increase the accessibility, acceptability, and the quality of health services. The law is also intended to restrain increases in the cost of health services, and to prevent unnecessary duplication of health resources.

The federal legislation calls for the creation of health service areas throughout the country, and for a network of health systems agencies (HSA) to carry out health planning and development within the service areas. Governor Castro has designated four health service areas for Arizona.

The Act also ties the existing medical facilities construction program (Hill-Harris) more closely to health planning and national health priorities. Specifically, construction funding will be more closely tied to alternatives to hospitalization such as ambulatory care, home health services and the provision of primary care services for the medically underserved, especially those in rural or economically depressed areas.

In carrying out their mandated responsibilities, the health systems agencies will prepare and implement health plans designed to meet the above-described goals in Public Law 93-641. As a part of this process, the HSAs will provide either technical assistance and/or limited financial assistance to people seeking to implement provisions of the plans. They will coordinate activities with PSROs and other appropriate planning and regulatory entities.

The legislation also assigns to the HSAs the responsibility for the review and approval or disapproval of applications for federal funds for certain health programs within the health service area.

Related HSA functions include assisting the state in making findings as to the need for new institutional health services proposed for the area and in reviewing existing institutional health services offered with respect to the appropriateness of such services. Annually the HSA will recommend to the state projects for the modernization, construction and conversion of medical facilities in the area.

The health planning law also calls for the designation of a state health planning and development agency; in Arizona the Department of Health Services will fulfill this function. The state agency will be advised by a Statewide Health Coordinating Council (SHCC) appointed by the Governor. Of the total membership, 60 percent must be from the state's HSAs, and a majority must be consumers.

My firm conviction that physicians must actively take part in health planning is reflected in the legislation. Public Law 93-641 states that "Since the health care provider is one of the most important participants in any health care delivery system, health policy must address the legitimate needs and concerns of the provider if it is to achieve meaningful results and, thus, it is imperative that the provider be encouraged to play an active role in developing health policy at all levels."

AM *Report on the Housestaff Section*

RATIONALE FOR GOVERNMENT INTERVENTION IN HEALTH CARE

HALLEY S. FAUST, M.D.*

The following is a personal viewpoint and does not necessarily reflect the position of the ArMA Housestaff Section.

Following a happily agreed to computer's decision of my displacement to Phoenix I have encountered a never-ending call of conservatism among medical professionals. The most frequent response of these conserving individuals is the shaking chill noticed when one mentions "government." No matter the context, no matter the emphasis, "government" makes a pneumonic crisis appear benign.

Though I too dislike government intervention which invariably is bureaucratically inefficient, there is a strong rationale for justifying its occurrence. This justification takes the form of the Socratian ". . . a state comes into existence because no individual is self-sufficient; we all have many needs."[1] To be responsible, a government must be responsive to those needs. With an appropriate social stimulus comes a governmental response—whether for the better or worse the intent is to meet a need of society. Needs of society develop priorities; for example, guaranteed food and clothing have traditionally come before a dishwasher in every home; education has been a guarantee for many years now.

In the field of health care we meet a societal demand: most facets of contemporary society have deemed worthy an attempt to provide a minimum standard of health care accessibility for all citizens. Our government has already made it public policy that "the achievement of equal access to quality health care at a reasonable cost is a priority of the Federal Government."[2] The fact that this passed the Congress makes us assume that this action is not a grossly grassroots unpopular one (except within the medical profession).

For the government to make health care access a priority does not necessarily mean that up to this time the medical profession has done a poor job in health care. However, it does mean that society's needs are not being met *now*. The American people are beginning to feel that regulation of health care services may be better met by consumers rather than providers.

Private medicine has been justified as being "private" for many years on various bases.[3] The physician may feel that any form of guaranteed right to health care access may jeopardize the doctor-patient relationship. The physician may feel a loss of autonomy, an inability to fully dictate medical practice on his own terms. But in the same way, the average citizen may feel that such close control of health care services by that same profession which supplies the services results in a decreased autonomy for the citizen in terms of his/her inability to choose the health care found most comfortable and economic to his/her own needs. The same right of autonomy the physician claims has resulted in a lack of autonomy in the right to health care of those being served.[4] As B. B. Page states in his "Who Owns the Professions?"

> Each (profession) insists on 'its' expertise, autonomy, and prerogatives; each seeks to make or influence the major policy decision in 'its' field; each develops interests, needs, and persepctives shaped within and oriented toward the profession itself so that it is often difficult for its members to apply their knowledge in ways likely to meet the interests, needs, and perspectives of society or of individual clients or patients.[5]

There are thirty million people under age 65 in the United States who do not have private insurance or public assistance in meeting the costs of health care. Of those who do have assistance most policies they own exclude preventive care, ambulatory care, prescription drugs, and medical devices. Many exclude pre-existing conditions. What terms are left in these policies are woefully inadequate considering today's economic health care burdens.[6]

There is a distinct difference between guaranteeing health care and guaranteeing health; the former is established through a rearrangement of resources, the latter is a factor of personal care and constitution. If following government's rearranging resources and guaranteeing health care we fear a glut of indignant patients demanding their care, then perhaps our emphasis ought to take a turn toward educating the public on the nature of health and the appropriate indications for utilization of new services. If we fear a substantial economic burden on ourselves and our families because of new government intervention and regulation, then perhaps we need to turn toward re-educating our personal value systems.[7] Jean Jacques Rousseau nicely summed up these considerations when he wrote,

> each individual, as a man, may have a particular will contrary or dissimilar to the general will which he has as a citizen . . . his absolute and naturally independent existence may make him look upon what he owes to the common cause as a gratuitous contribution, the loss of which will do less harm to others than the payment of it is burdensome to himself . . . whoever refuses to obey the general will shall be compelled to do so by the whole body.[8]

This speaks well for what is currently occurring *to* medicine. Society has determined that for too long the profession has had a will "contrary or dissimilar to the general will" and now we will be compelled to obey the general will.

I have not attempted to analyze the various (non)attributes of the current health care system,[9] nor have I considered the potentially best modes of government intervention. Instead I have attempted to give some rationale for government intervention in the field of health care delivery. I doubt, however, if the physician's frenetic fever generated following the shudder-oid response to "government" can be broken anytime soon.

REFERENCES

1. The Republic of Plato. London: Oxford University Press, 1973. p. 55.
2. Public Law 93-641. "National Health Planning and Resources Development Act of 1974."
3. To name a few, these frequently stated justifications include "the doctor-patient relationship is inviolable . . . the government can't tell me how to best treat my patient . . . it's been this way for years and must be good." There are some substantial truths to parts of these statements, but they avoid the issue of necessity for health care access, which does not necessarily imply interruption of the free interaction between the physician and patient.
4. "What right to health care exists?" you may ask. Rights are only available when granted by some authority. In this case the right to health care is deemed valid by our Federal Government, who, of course, guarantees our other rights in the social contract system.
5. Hastings Center Report 5(5):7-8. October, 1975.
6. "About one half of private policies limit total benefits payable in a lifetime to $10,000 or less, and almost two-thirds of current policies limit hospitalizations to sixty days or less." Forward Plan for Health FY 1977-81. Department of Health, Education, and Welfare Publication #76-50024. June, 1975. pp. 162-3.
7. In government's view: If to improve resources (health care) to a large segment of our population we need to diminish (monetary resources) to a much smaller population (physicians) then it seems we are justified in so doing.

   "It is monstrous if an individual doctor thinks like a budget officer when he cares for his patient in need; but it is chaotic and incoherent if budget officers and voters making general policy think like doctors at the bedside." from C. Fried, "Equality and rights in medical care." Hastings Center Report 6(1):29-34. February, 1976.
8. Rousseau, Jean Jacques. The Social Contract. New York: E. P. Dutton, 1950. pp. 17-18.
9. For a provocative critique on contemporary health and health care see Illich, Ivan. Medical Nemesis—the Expropriation of Health. New York: Pantheon Books, 1976.

Editorial

CAN MEDICINE SURVIVE THE ADVERSARY SYSTEM?

SHERMAN M. MELLINKOFF, M.D.

All of us have persisted, more than once, in using a clearly unworkable approach to an increasingly frustrating problem. The wrong key that fits halfway into a lock is a common example. Even great physicists have tried for a lifetime to fit facts into the wrong theory. But when it comes to applying the wrong method to an unsolved puzzle, surely mathematicians, engineers, and physicists must take a back seat to those of us embroiled in sociology.

In the history of the human race the general tendency is to select an irrational theory and use it relentlessly. The Crusades lasted 200 years, accomplishing quite a bit of misery on a wide scale. Religious feuding is still ablaze in the "Holy Lands." The "Thirty Years' War," which began in the sixteenth century A.D., persists in blasting buildings and vehicles, some of which contain people, in Ireland and England.

Those of us concerned with medicine, however, need not yield to kings and other rulers, nor to generals, legislative bodies, and guerrilla leaders, as strong adherents of the wrong-key principle. Let the fates rest assured that we are doing our part.

Take the adversary system. It works fairly well in law and politics. At least other systems are much worse. Impeaching or even electing a president may be laborious, but not nearly as offensive as being saddled with a bad commissar who need not worry about re-election, or, if so, by no more than a few handpicked voters. Similarly, court battles, whether civil or criminal, are expensive and time-consuming, but not as annoying as decision by fiat, especially to the person whom the decision is against.

It is no wonder, then, that the adversary system has been fastened onto medicine. In keeping with the wrong-key principle, however, we are using the adversary system with more and more zeal the clearer it becomes that we are making a mistake.

Consider, for example, the adversary system and risky medical or surgical therapy. Remedies for previously fatal diseases have risen logarithmically in recent decades. Inevitably the treatments involve risks. Thus, subacute bacterial endocarditis, formerly 100 percent lethal, can now often be cured, but only by using drugs that sometimes have adverse side effects. The side effects afford more opportunities for litigation, particularly when this popular sport is profitable.

Before surgery of the heart was feasible, many patients were by their illnesses sentenced to early death. Many can now be cured by operations that carry a mortality rate considerably higher than a week or two in bed. Suppose one of these patients suffers cardiac arrest for long enough to damage his brain and requires prolonged medical and nursing care. It may well be that the surgeon, the anesthesiologist, the nurses, and the hospital are all blameless. But so is the patient, and his family, and lacking catastrophic health insurance, which everyone should have, it seems to me, the patient's family and either the first or second lawyer they consult will decide to sue for malpractice. How else can they finance the care of their loved one? The adversary system takes over, with a fair amount of money lost in overhead. Depositions, court costs, rising insurance premiums, lawyers' fees, and possibly a tendency towards aggravation of peptic ulcers, most forms of nocturnal pruritus, hypertension, and angina pectoris—the doctor experiences all these instead of an expert, dispassionate judgment of what medical care the patient should have under the circumstances.

The anesthesiologist, for one, in this case may well have learned something deleterious about "informed consent." The lesson is clear long before the plaintiff's lawyer has finished cross-examining the hapless doctor on a day he had planned to be in the operating room. What he has learned he applies to the next patient he sees, an apprehensive old man who needs a bowel resection for cancer. The poor patient has just barely decided to set aside his fears on advice of the doctor who told him the operation would not be much more of a risk than driving to New York from San Diego, whereas without operation chances of recovery are nearly zilch. But now the anesthesiologist, sensitized by the adversary system in all its majesty, feels obliged to tell the old man that anesthesia itself carries certain hazards, more so for the aged.

"What hazards?" the old man asks.

"Atelectasis, pneumonia, cardiac arrest, aspiration of gastric contents, liver disease . . . "

"Wait a minute," the old man pleads, "what is atelectasis? I never heard of it before."

In ten minutes conversation the anesthesiologist has insured that if the patient still consents to surgery he will be nervous enough to augment a variety of dangers, possibly bleeding postoperatively from a "stress ulcer." But no lawyer can correctly claim the anesthesiologist failed to obtain truly "informed consent."

The doctor who diagnosed the cancer in the first place will also have profited greatly from studying his role in the adversary system of medical practice. Here was this 50-year-old man complaining of weakness and roaring in the ears, both explained easily by the hematocrit of 20 and the wide pulse pressure of anemia. Physical examination, remarkable only for pallor and a vague mass near McBurney's point, leads to a barium enema and X rays outlining a carcinoma of the cecum. Instead of feeling satisfied that he has fairly expeditiously identified a possibly remediable illness, fatal without surgery, the doctor by no means looks at once for a good surgeon. How about that roaring in the ears? Better have a complete ENT work-up, and, of course, a bone and liver scan in search of metastases, and an alkaline phosphatase and enough other "chemistries" to find at least one outside normal limits. Maybe the alkaline phosphatase is a bit high from a small irrelevant patch of asymptomatic Paget's disease. Never mind. There must be no expense, no inconvenience, and no confusion spared to avoid some lawyer's future charge of negligence.

"Did you subject this man to a bowel resection, which led to death from a pulmonary embolus, without first making sure he did not have bone metastases?" This stentorian question echoes in the doctor's hypothalamus and spurs him on to be as inflationary and alarming as possible.

It might be possible for doctors and medical educators to arrest this spiral of fear, this erosion of fiduciary trust between healer and patient. Remedies more rational than the adversary system to safeguard patients and their woes could be evolved. But fortunately for the wrong-key principle this eventuality may be posponed almost indefinitely provided we all exercise a maximum of energy compressed into a minimum of wisdom. After all, *anyone* can sue. The lawyers and the law are guardians of that right. And, happily, even the most aggressive lawyer or slothful legislator who happens to need a good doctor can find one somewhere, thus assuring that no one learns a personalized lesson.

Redeemingly, however, human beings, as Mark Twain observed of Wagner's music, are not as bad as they sound. Mixed with avarice, fear, and litigious hostility, are kindness, conscience and reason. Otherwise medical practice would not be as excellent as it so often is. No matter what the lawyers and the small print say about informed consent, most doctors would rather face a subpoena than to develop insomnia remembering what they should have done, but did not do, for a dead patient. The Hippocratic law is older and more durable than many newer ones. Would that review mechanisms were as effective as the honest questions of an idealistic resident!

The adversary system of weighing medical risks must stop short of malpractice premiums that would make old King Midas sigh and Hippocrates become a full-time farmer. And so it will. As Abba Eban has correctly observed, "Both men and nations do behave wisely once they have exhausted all other alternatives."

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WARNINGS. Since drowsiness may, on occasion, occur with use of this drug, patients should be warned of this possibility and cautioned against driving a car or operating dangerous machinery.

Usage in Children: Clinical studies establishing safety and effectiveness in children have not been done; therefore, usage is not recommended in the pediatric age group.

Usage in Pregnancy: See "Contraindications."

ADVERSE REACTIONS. Drowsiness, dry mouth and, on rare occasions, blurred vision have been reported.

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ARIZONA MEDICAL ASSOCIATION
FOUNDATION, INC.
funded by
ROBERT WOOD JOHNSON FOUNDATION
GRANT NO. 1636

FIRST ANNUAL REPORT
July 31, 1976

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I

Introduction

This is the first annual report of the Arizona Perinatal Program, a program initiated with support from the Robert Wood Johnson Foundation, Grant No. 1636. It covers the period August 1, 1975 through July 31, 1976. The report is presented in format similar to the one used in the 1975-76 Program Plan, which simplifies measurement of the degree of attainment of the year's operational objectives. Concise summaries of actions considered important to fulfill specific goals are supplemented, when appropriate, by appended detailed narratives.

This type of presentation may not clearly reflect the interdependency and congruence of the several program components. It should be pointed out, therefore, that attainment of Program aims will ultimately constitute a system of regional perinatal care which should positively impact on Arizona's perinatal morbidity and mortality. This should also serve as a practical model for superior health care to mothers and infants in other regions. It follows that this will raise the quality of life at its beginning.

Much staff time has been spent during this first year developing plans under the leadership of a strong Executive Committee and seven task groups composed of many talented men and women. Simultaneously, substantial actions have occurred and, although objective evaluation of these important actions will not be available for some months, observations from preliminary and incomplete data give cause for optimism that the next 18 months will see concepts of the Arizona Perinatal Program firmly established as part of Arizona's total care system.

Significant progress has been made toward accomplishing most of the first year's objectives. The way in which Program plans extend into the coming year is outlined in Part III, "Program Plan 1976-77."

Mission

To improve the outcome of pregnancies in Arizona by assuring the accessibility of high-quality perinatal and related health care which matches the level of need.

II
SUMMARY OF ACTIVITIES
1975-76

Development of Data Base

In early October 1975, a Task Group of the Arizona Perinatal Program (APP) undertook development of a uniform clinical data base system which incorporates an assessment of perinatal risk. The objective of the system is to identify problems during the perinatal period which constitute a relative risk to both mothers and infants by screening all pregnant women early in pregnancy and by determining the degree of risk, using this as a predictor of outcome, which would be helpful in the appropriate use of regional care facilities and resources.

The Task Group adhered to the principle that flow of recorded clinical observations on the data base should follow established, logical thought patterns of trained, experienced health professionals. The computer-compatible data sheets, which also serve as paper clinical records, exceed requirements of the minimal clinical data set suggested by Doctors Shapiro, Krischer, et al, of Johns Hopkins University.

Pre-tests conducted at a small number of sites were completed by July 31, 1976; final drafts will be available for review after the present revision has been further tested. The initial field tests of the antenatal record have been well accepted in physicians offices, secondary and tertiary centers.

Data System

The objective of the system is to record, collect and store relevant data on perinatal events which might influence the management and outcome of pregnancy. This requires a system for transmittal of data from all participating care centers to a central computer where the data may be stored and easily retrieved for appropriate analyses.

Such a system was under way with the installation at St. Joseph's Hospital and Medical Center of a Data General Eclipse Computer, Model S 200. Completed July 19, 1976, installation of this computer and development of appropriate programs will allow maintenance of all patient data in an alphabetical structure which will avoid duplication of information, even in cases where a patient is transferred between facilities, both of which are entering data into the Arizona Perinatal Program Data System.

The computer model in use contains 40-K units of core storage, sufficient interface equipment for simultaneous communications with 17 terminals, disc storage of 22 Diva discs with a total capacity of 100 million characters of data and a Pertec tape drive unit and controller. The initial programming is in the Meditech MIIS (MUMPS) language. The system is expected to be functional by mid-August 1976.

Programming for entry and retrieval of data from the data sheets ("Admission Status Record," "Labor-Delivery" and "Neonatal Data I") will start in late August 1976. Subsequent data sheets should be ready for entry within one week after the final form of the data sheet is determined.

All data entries from St. Joseph's Hospital Department of Reproductive Medicine (DORM) will be done on a daily basis using internal, hard-wired terminals. Outside hospitals, such as Maricopa County General Hospital with 4,000+ deliveries per year, will need to enter approximately 16 patients per working day into the computer. Including all six data bases now under test, it is estimated an experienced clerk should spend about two to three hours per day entering data on the 16 patients. Data sheets from smaller units will, with one or two exceptions, be "batched" weekly or monthly and mailed to APP headquarters for entry into the computer system, either by a "dial-up" telephone line or directly from a terminal at St. Joseph's Hospital. It is estimated by consultants that one full-time data clerk would be capable of entering data from approximately 10,000 patients per year.

Protocols for protection and safety of tape copies will be developed by Jerry Isaman, M.S., staff Biomedical Specialist, and Don Gall, Sc.D., Bioengineering Consultant. Mr. Isaman will be responsible for procedures for transmittal of data to the Johns Hopkins evaluation group.

Graded Levels of Care

The plan for development of graded care facilities is an adaptation of two documents: "Toward Improving the Outcome of Pregnancy," published by the National Foundation/March of Dimes in the Spring of 1976; and the preliminary recommendations of the Committee on Perinatal Health, as summarized by George M. Ryan, Jr., M.D.[1]

The small Task Group responsible for the Arizona Perinatal Program (APP) recommendations developed the strategy of "a moveable feast," with the Executive Director and one or two colleagues visiting principal hospitals in eight of the State's twelve rural counties over a period of nine months. This process was designed to inform community hospital personnel at all levels about the concept of regional perinatal care.

The Ryan article was sent to the chief-of-staff of each hospital two months prior to a scheduled meeting. As it evolved, draft copies of the APP plan were also distributed in advance of the meetings. Following each session, critical comments were solicited and, when appropriate, incorporated in the next revision.

It was pointed out that the sessions were not for the purpose of recommending the plan as an established set of guidelines, but rather to seek advice on how best to adapt carefully developed recommendations from other parts of the country to a region with unique demographic, geographic, cultural, medical and political characteristics. It was also stressed that the recommendations are a platform upon which the institutions can build superior quality perinatal care. The third draft of the recommendations is part of the unabridged annual report (Appendix #1*).

As a followup to the orientation sessions, requests have been received from several institutions which wish to become part of APP's system of Primary, Secondary and Tertiary Care Centers. Careful consideration has been given to each request, taking into account whether or not the institution meets the criteria established for that level of care.

The following hospitals have contracted with APP to be the Tertiary Care Centers: St. Joseph's Hospital and Medical Center, Phoenix, and University of Arizona Hospital, AMC, Tucson. Flagstaff Community Hospital requested to be accepted as a Secondary Care Center, and has been accepted at the direction of the Executive Committee to serve as the prototype for Secondary Care Centers. Geographic location and potential patient area make this a particularly appropriate center. Phoenix Indian Medical Center is being considered as an unfunded Secondary Care Center. Whiteriver Apache Health Facility (Indian Health Service) and Northern Cochise Community Hospital at Willcox have both indicated their desire to become Primary Care Centers and will be considered for acceptance.

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Arizona Clinical Perinatal Center Coordinators:
Phoenix – St. Joseph's Hospital
Raymond J. Jennett, M.D.
Stephen M. Lantz, M.D.
Donald J. Ziehm, M.D.
Tucson – University Hospital
Harlan R. Giles, M.D.

It is expected that in the near future, a direct line to the Clinical Perinatal Center Coordinators in both Phoenix and Tucson will be available. Until that time, the following procedure will be used.

FOR CONSULTATION AND/OR TRANSPORT OF A HIGH-RISK MATERNITY PATIENT:

- Contact a CPCC at a Tertiary Care Center.
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  - Your name and phone number.
  - Your location.
  - That you wish to speak to the Clinical Perinatal Center Coordinator on call—(Phoenix – St. Joseph's Hospital or Tucson – University Hospital)

  The physician on call will be in contact with you within a few minutes.

THE TELEPHONE NUMBERS TO CALL ARE:

PHOENIX - 258-8331
(Maricopa County Medical Directory)
TUCSON – Weekdays: 8 a.m. to 5 p.m.
882-6635 or 882-6636
(Arizona Medical Center Perinatal Office)
TUCSON – Nights and Weekends
882-6300
(University Hospital - Main Switchboard)

For other information write or call: The Arizona Perinatal Program, 810 W. Bethany Home Rd., Phoenix, Az. 85013, (602) 246-8421.

For your personal convenience, clip and carry this card.

ARIZONA PERINATAL CENTERS:

Phoenix – 258-8331 (Maricopa Co. Medical Directory)
Tucson – Weekdays - 8 to 5 – 882-6635 or 882-6636
(Arizona Medical Center-Perinatal Office)
Tucson – Evenings, Weekends & Holidays – 882-6300
(University Hospital - Main Number)

Give following information: Your name & phone number, your location. Ask to talk with the Clinical Perinatal Center Coordinator on call regarding a high-risk pregnancy patient.

Figure 1

Present budget limitations dictated the amount of funds available to participating Tertiary and Secondary Care Centers for partial support of patient care. This consideration influenced the decision that two Tertiary Care Centers and one Secondar Care Center would receive funding for patient care and maternal transport in the present and succeeding fiscal year.

Access to Care: Communication System

Since May 1, 1976 a single telephone number has been abailable to physicians in the outlying areas of the State who required consultation with a perinatologist or a neonatologist in Phoenix. This was provided by the Medical Directory, Maricopa County Medical Society. Different numbers have been used for the Tertiary Care Center in Tucson. These telephone numbers and all relevant information and instructions were supplied on a card which was mailed to all Arizona hospitals and physicians active in obstetric and pediatric practice. See Figure 1.

A more flexible system has been developed which will link any Arizona health professional quickly to either a perinatologist or a neonatologist in Phoenix or Tucson. A prototype of the new system was tested in Tucson for six weeks beginning in June 1976; the total system will be operational in Tucson by the time this report is completed and the Phoenix system will be in operation soon afterward. The system utilizes General Communication services (a nationwide mobile telephone company) and individual 5-channel VHF mobile two-way radio transceivers. Any health professional in Arizona can thereby be linked with the "on call" member of a group under the guidance of the Clinical Perinatal Center Coordinator (CPCC) by means of the radio system. The single number for this system in Tucson is (602) 624-6646 and in Phoenix (602) 253-6143.

The Arizona Perinatal Program (APP) has made the capital investment in mobile equipment, installation and licensing fees, which totals approximately $6,000 for each Tertiary Care Center. Monthly use fees will be approximately $30.00 for each transceiver in use, and the two Tertiary Care Centers will be requested to assume these recurring charges. A transitional period of about twelve weeks is planned for the changeover from the existing system to the new system. During this 12-weeks period, the new telephone numbers and a brief descriptive brochure on how to use the system will be given wide distribution to all potential users.

Access to Non-routine Laboratory Services

There have been delays, beyond the control of the APP staff, in development of the transportation network for patients requiring non-routine laboratory procedures. These delays necessitate a report of "no progress" at this time. This does not mean laboratory facilities in the two Tertiary Care Centers are not being utilized, but only that the services have not yet been systemized or widely publicized. It is expected the system proposed in the APP Program Plan for 1975-76 will be fully operational by January 1, 1977.

Education

The goal of this operational component is to have well-trained health professionals available, in terms of both numbers and geographic distribution, to provide high quality perinatal care to all high-risk mothers and infants in Arizona. In conjunction, an educational program is required that will provide potential patients and the general public with information about high-risk pregnancies, the importance of perinatal care and care between pregnancies, and the location and means of access to perinatal services in the State.

Education Task Group

Although budget constraints for educational activities have limited the progress made towrd achieving the stated goal, significant accomplishments by a multi-disciplinary group can be reported. During this year, an ad hoc Education Task Group was formed to develop a statewide plan for the coordination of education in perinatal care directed to health professionals, patients and the general public.

The Task Group Chairman appointed three subcommittees to complete necessary tasks and submit recommendations to the group as a whole. The subcommittees were: Professional Education, Patient Education, and Public Education. Subsequent to these appointments, a committee of the Advisory Council assumed the responsibilities of the Public Education Subcommittee so this group did not meet.

Both the Professional Education Subcommittee and the Patient Education Subcommittee, after independent and joint meetings over a period of months, submitted recommendations which were subsequently presented to the Executive Committee for consideration. Both subcommittees recommended that the APP investigate various sources of funding to support the educational program and employ a full-time staff person as Education Director, who would coordinate and assist in planning educational programs for perinatal health professionals and patients throughout the State.

Other recommendations from the subcommittee emphasized the need for evaluation of all educational programs; assurance that continuing education credits were offered to professional participants; initiation and development of patient education programs aimed at reducing the risk of premature labor, emphasizing the importance of early prenatal care, and recognition of warning signs and symptoms of preeclamptic toxemia.

Inventory of Continuing Education

Information about continuing education programs in perinatal care, offered statewide during 1975-76, was obtained from the Arizona Nurses Association and the Arizona Medical Association. A list of the programs is included in* the unabridged annual report (Appendix #3).

Proposed Educational Plan

Based primarily on the recommendations and work of the Education Task Group, a comprehensive educational plan was developed for Arizona which includes objectives, activities and an estimated time schedule for completing activities. (The Plan is Appendix #2 of the unabridged annual report.) The three major areas of activity for the APP described in the Plan are briefly as follows:

1) Assess training needs which will identify health personnel involved in all aspects of perinatal care and identify the types and levels of training needed for existing and potential staff.
2) Develop a plan to coordinate existing professional education and training functions in order to reduce

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[1] Ryan, G.M., Jr., M.D.: "Toward improving the Outcome of Pregnancy: Recommendations for the Regional Development of Perinatal Health Services." J. Obst. & Gynec. 46: pp 375 ff (Oct 1975)

*All Appendices are on file in the Arizona Perinatal Program Office, Phoenix, and copies are available upon request.

duplication of programs and coordinate cooperative programs among various institutions.

3) Evaluate and followup education and training programs to determine the effectiveness of training and identify the needs for qualitative and quantitative changes in training as programs and technologies develop.

A major educational emphasis will be to develop and strengthen the team approach to perinatal health care. In this approach, the patient and her family will receive special attention as members of the health team which includes physicians, hospital administrators, nurses, aides, social workers, nutritionists, and others.

Grant Application

The educational plan was incorporated into a grant application to the National Foundation/March of Dimes (NF/MOD) to support a program which primarily aims at coordination and extension of numerous educational efforts being undertaken by existing institutions and individuals. Total amount of the grant request is $123,000 over a two-year period.

Newsletter

The first issue of the *Arizona Perinatal Program News* was published in July 1976. This newsletter is primarily aimed at health professionals and will be published bi-monthly. The editor and editorial board are members of the APP Advisory Council. Copies of the editorial policy and the first issue are in the unabridged annual report (Appendix #4).

Other Education and Information Activities

An exhibit which shows the basic structure and purpose of the APP was designed and displayed at the Arizona Medical Association 85th Annual Meeting in April 1976. It was also shown in a large Phoenix shopping center as part of a public education effort by the Arizona Department of Economic Security, Bureau of Mental Retardation, and at a Fetal Monitoring Workshop at St. Joseph's Hospital in June. Attractive and colorful, the exhibit's modular construction, the ease with which the message panels can be changed, and the addition of various audio-visual modes are features which assure long-term usefulness of the unit for a variety of audiences.

Maternal Transport System and Protocol

With a goal of providing all high-risk maternity patients a coordinated and efficient transport system for their transfer to the appropriate care center, an ad hoc Task Group was formed to develop policies and procedures for such transports.

The document produced by the Task Group was expanded by the APP staff and carefully reviewed by the appropriate professionals. Included are procedures for requesting consultation and possible transport of high-risk maternity patients, professional and technical qualifications of the transport team, and lists of necessary transport equipment. When in final form, the protocol will be distributed to referring physicians and hospitals, as well as Tertiary Care Centers. The protocol is included in the unabridged annual report (Appendix #5).

Transport Patients

From December 1, 1975 through June 30, 1976 there were 126 high-risk maternity patients transported to the Tertiary Care Center in Tucson, University Hospital, from approximately 21 outlying communities and hospitals in metropolitan Tucson. These patients spent a total of 632 days or an average of five days per patient in the hospital.

TABLE ONE

MATERNAL TRANSPORT PATIENTS

Month/Year	Number of Patients University Hosp.	 St. Joseph's Hosp.
Jan. 1975	11	2
Feb.	9	1
Mar.	11	5
Apr.	5	3
May	16	5
June	12	5
July	10	8
Aug.	12	7
Sep.	13	1
Oct.	15	2
Nov. 1975	15	7
MEAN for period	11.7	4.2
Dec. 1975	11	6
Jan. 1976	18	6
Feb.	13	7
Mar.	25	8
Apr.	18	10
May	23	10
June	18	16
MEAN for period	18.0	9.0

Since start of Program, mean number of patients at University Hospital increased 53.8%; at St. Joseph's, 114.0%.

Source: Dept. of Ob/Gyn, College of Medicine, Univ. of Ariz., Tucson; Dept. of Reproductive Medicine, St. Joseph's Hosp. & Med. Cntr., Phoenix. July 1976.

During the same period, 63 patients were admitted to St. Joseph's Hospital in Phoenix as high-risk maternity patients for a total of 405 days for an average hospital stay of 6.4 days. As can be noted in Table One, the number of patients transported to both Tertiary Care Centers has been increasing since January 1975.

Figure 2 and Table Two show that patients are being transported to Tertiary Care Centers from communities throughout the State as well as suburban areas around Phoenix and Tucson.

Followup of High-Risk Patients

In order to provide all high-risk mothers and infants with appropriate followup care, APP established two objectives:

1) development of guidelines for followup; and 2) development of similar guides for identification of correctable defects and health problems. The target date for completion of these documents was set for January 1, 1977, as indicated in the 1975-76 Program Plan (p.58).

Implementation of a protocol for neonatal followup will be shared by the Bureau of Maternal and Child Health, Arizona Department of Health Service. The Bureau has had an established protocol since 1971, although it has not been consistently implemented nor results regularly reported due mainly to manpower shortages. Revised procedures were published as of July 1, 1976 (Appendix #6). Examinations are done at the time of discharge from the Neonatal Intensive

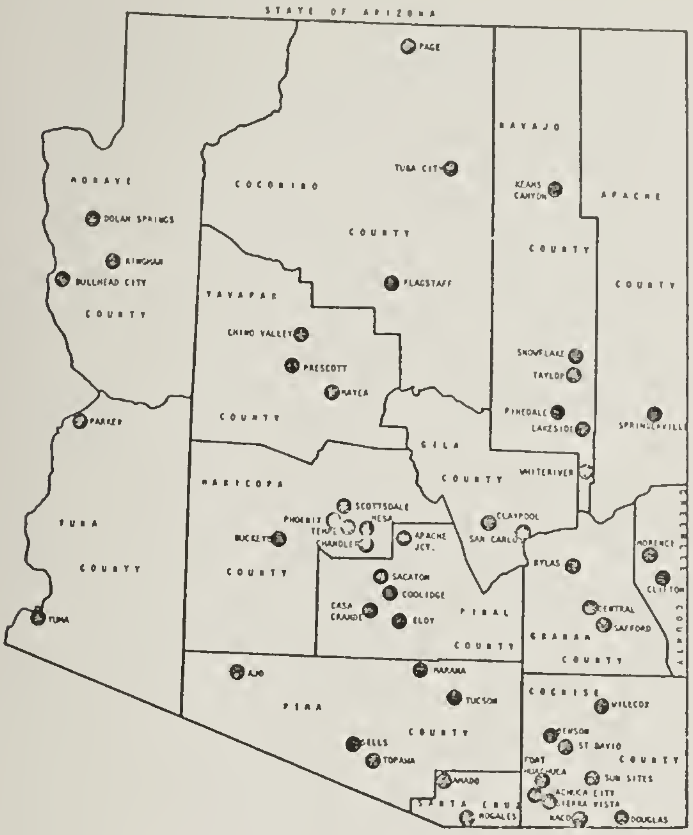

Figure 2

A map of communities from which patients transported to tertiary care centers at Phoenix and Tucson. December 1975 through June 1976.

Care Unit (NICU), and followup examinations follow at six months, one year, and two years of age (Appendix #6).

The followup caseload for fiscal year 1975-76 for State supported NICU nurseries was 1,243; 75% of these infants were born to residents of the two metropolitan counties (53% in Maricopa County; 22% in Pima County). The Bureau supplies Maricopa County a team of two Public Health Nurses skilled in the followup procedures. In the other counties, followup is presently dependent upon local health depatment staff personnel, who are already heavily committed to a variety of other programs.

The Newborn Followup Program directed by Elsa J. Sell, M.D., Department of Pediatrics, University of Arizona, has been underway since November 1975. The initial proposal is appended to the APP 1975-76 Program Plan, on file at the Robert Wood Johnson Foundation, Princeton, New Jersey, and at Johns Hopkins University, Baltimore, Maryland, and has been reviewed with Janet Hardy, M.D. The proposal precis states . . . .

> "(We) will attempt to identify certain characteristics of a pilot group of infants who have survived neonatal intensive care in Arizona. The incidence of neurologic, developmental and psychological abnormalities will be defined. Risk factors which may be predictive of future outcome will be assessed. Finally, new research tools will be utilized to attempt earlier discovery of abnormal outcome in neonatal intensive care survivors. The pilot group, for the first year, will contain approximately 140 patients; additional patients will be added in subsequent years of the project."

A copy of the first progress report from Doctor Sell is included in the unabridged annual report (Appendix #7). We believe this project is proceeding well with fulfillment of the stated objectives, although there is a lag in numbers of patients involved. It is obviously too early to draw any other conclusions at this time.

Table Two

MATERNAL TRANSPORTS BY COUNTY

December 1975 thru June 1976

County Name	No. of Patients
APACHE	1
COCHISE	26
COCONINO	4
GILA	2
GRAHAM	6
GREENLEE	7
MARICOPA	42
MOHAVE	4
NAVAJO	6
PIMA	68
PINAL	11
SANTA CRUZ	4
YAVAPAI	5
YUMA	2
	188

Source: Dept. of Ob/Gyn, College of Medicine, Univ. of Arizona, Tucson; and Dept. of Reproductive Medicine, St. Joseph's Hospital and Medical Center, Phoenix. July 1976.

The principal achievement of the project has been establishment of a research team under the leadership of Doctor Sell and overall research supervision of Grant Morrow, M.D., Professor and Associate Head, Department of Pediatrics.

The main problems relate to a precise dependency on the APP for total financial support. The Program cannot at this time assure support beyond that committed in our first budget, I.E., $40,000 per year. The APP staff is pledged to work with the Department of Pediatrics in searching for supplemental funds.

Administration: Planning

The first draft of the Program Plan for the APP was completed in March 1976. It is anticipated the Plan will be revised annually to provide direction to the APP, and the revision will include a summary of activities during the previous year.

The first Program Plan contains an historical account of the genesis of the APP, geographic and demographic profiles of the State, a summary of available relevant health indicators, a description of existing resources for providing perinatal care, and the initial operational goals and objectives for the Program. The Operations Review Committee of the APP Advisory Council reviewed the Plan and made suggestions for revision and modification in some areas. The recommended changes which were approved by the Executive Committee are included in the goals and objectives for fiscal year 1976-77.

Interim Governance

The Perinatal Planning Subcommittee of the Maternal and Child Health Care Committee, Arizona Medical Association (ArMA), served as the Interim Executive Committee for APP until the By-laws and Constitution were approved. This interim committee met five times during the first months of the Program's existence. The majority of the business focused on development of the By-laws and Constitution and establishment of policy direction for the Program.

The Maternal and Child Health Care Committee of ArMA served as the APP Interim Advisory Council. Their primary responsibilities included review and approval of the By-laws and Constitution and selection of the permanent Advisory Council.

Organization

It is the intent that the organization of the Arizona Perinatal Program (APP) will be structured to allow maximum participation by health professionals and consumers in planning and evaluation of the perinatal care system in Arizona. To comply with this intent, the APP By-laws and Constitution, as developed by the Interim Executive Committee and approved by the Interim Advisory Council, provide for an eight-member Executive Committee and a multi-disciplinary Advisory Council of up to 36 members with representation from various geographical areas of the State.

The permanent Advisory Council was selected from lists of Arizonans recommended by various organizations as candidates for position on the Council. In selection of members, consideration was given to geographical distribution as well as interest and previous experience in maternal and child health. Figure 3 shows the distribution of Executive Committee and Advisory Council members.

The eight-member permanent Executive Committee held four meetings from January 1976 through July 1976. The 31-member permanent Advisory Council met for the first time in November 1975. Officers were elected and the Council discussed the objectives of the APP and the broad perspective of regionalized perinatal care.

At the next meeting of the Advisory Council (March 1976), the standing committees were established and chairpersons appointed. The committees were: Communications, Liaison, Operations Review, and Resource Development. An executive editor and editorial board were also appointed. Subsequently committee deliberations have provided program direction.

Resource Development

The goal of this operational component is that resources required for continuation and expansion of the perinatal care system in Arizona will be fully developed by August 1, 1980.

The Resource Development Committee of the Advisory Council, activated in May 1976, is charged with responsibility for development of a long-range plan for creation of sustaining financial resources from private, State and Federal sources.

The major objectives of the Committee are: 1) to have current patient-care support integrated into the ADHS Bureau of Maternal and Child Health Programs, and 2) to establish an endowment trust sufficient to maintain an independent Arizona perinatal foundation which will serve as a center for limited but important support services for maternal and child health activities.

Grant support for development of regional perinatal education has been requested from the NF/MOD. Action on the grant application, forwarded to the Arizona Chapter NF/MOD in early May 1976, is not expected before September 1, 1976.

The Meade Johnson Company contributed $3,000 to the education component of the Program in May 1976.

The Arizona Perinatal Program applied for and received a one-year grant in the amount of $226,000 to continue an established maternal and infant care program on the Whiteriver Apache Indian Reservation. Effective July 1, 1976, the APP became the effective management contractor for what is known as "Project Apache." A detailed operations plan is on file in the executive offices to APP.

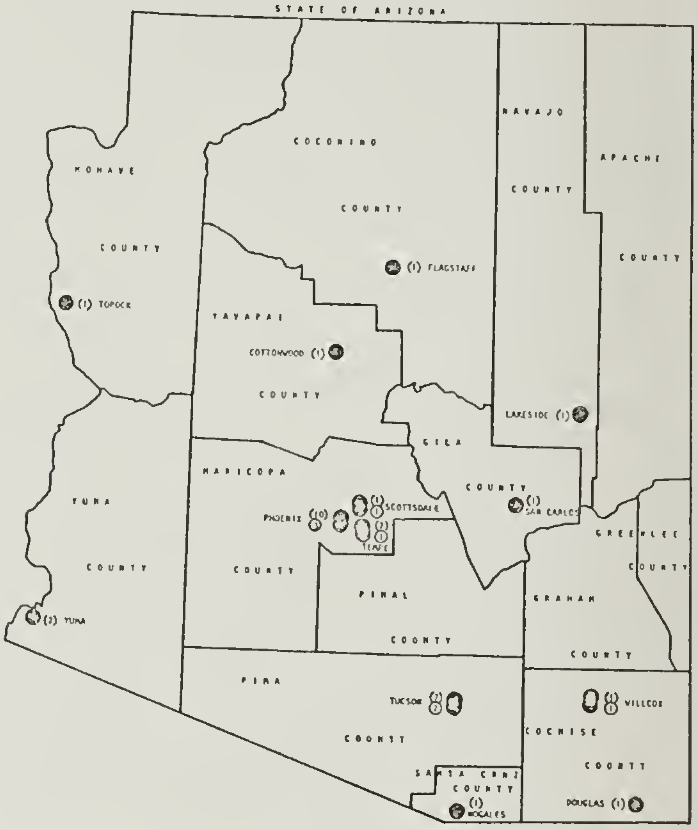

Figure 3

A map showing location and number of advisory council and Executive Committee members. Arizona Perinatal Program, 1976. Legend: ( ) Advisory Council members; 0 Executive Committee members.

Evaluation

The efficiency and effectiveness of the APP, as well as the impact on the health status of mothers and infants in Arizona, will be ascertained through the evaluation process shared with the Health Services Research and Development Center, Johns Hopkins University, Baltimore, Maryland.

Indicators of gross program impact, that is, fetal, perinatal, infant and maternal mortality rates from 1968 through 1975, have been tabulated and made available to the Johns Hopkins group as part of the APP Program Plan. A list of the tables, data for which was obtained from the Bureau of Vital Statistics, ADHS, is Appendix #9 of the unabridged annual report.

The method for transmitting tapes has been worked out by Mr. Jerry Isaman and matched birth-death certificates on tapes, 1968 through 1975, have been forwarded to Baltimore. Arrangements have been made to routinely forward such tapes as they are completed.

In a given program population, those indicators which have demonstrated good correlation with selected impact indicators tend to be little affected by qualitative or quantitative variations. The following are to be routinely reported for mother and infant by ethnicity:

a) percent of live-born infants with low birth weight (2500 grams or less);
b) percent live births with short gestation (35 weeks or less);

c) percent of women with pregnancy intervals of less than 12 months;
d) percent of women admitted to prenatal care during the first trimester of pregnancy;
e) percent of women referred for between pregnancies care of any type;
f) median time of comprehensive infant followup after discharge from the hospital.

Data on the first four of the above six indicators are regularly available form the Vital Records Section, ADHS, and are currently being tabulated. The last two are to be taken from the maternal and infant followup studies. Tabulations will be forwarded to the Johns Hopkins group as they become available.

The survey of health professionals referred to in the Program Plan has not been started. It is anticipated these knowledge, attitude and program-use questionnaires will be initiated by March 1, 1977 or soon thereafter.

No comprehensive plan for neonatal morbidity studies has yet been developed. Special studies will be designed during the coming fiscal year as information from the medical data sheets and risk screens on maternity and neonatal patients is collected and stored in the central computer system. The APP staff anticipate an early meeting with Doctor Krischer of Johns Hopkins University will resolve matters of computer programming to develop specific correlations between clinical events recorded on the data sheets, including correlations of specific interventions with pregnancy outcome. Special programs which focus these data for immediate clinical management will be developed within the next twelve months.

Efficiency Indicators

A plan for studying cost-effectiveness mentioned in the 1975-76 program plan has not been developed. A single study on cost-effectiveness has been reported by Thomas Harris, M.D., Chairman of the APP Communications and Data System Committee. However, since this was an independent effort and Doctor Harris's conclusions were provisional, we have not included these data. The study may serve as a model for an expanded study to be started March 1, 1977.

Reports and Publications

Monthly financial reports prepared by the Fiscal Officer of the Arizona Perinatal Program are forwarded to the office of the Executive Director of APP. These reports are consolidated into an Annual Financial Report, included as Part IV of the unabridged annual report and summarized in Part IV of this document.

A preliminary report on certain aspects of the maternal transport patients is in preparation and will be submitted for publication to a professional journal following presentation at a regional obstetrical society meeting in October 1976.

In the coming fiscal year, narrative quarterly performance reports will be prepared by Staff and submitted to the Executive Committee. During the past year informal reports have been presented in summary at each Executive Committee meeting. These reports dealt principally with planning and policy matters and are part of the minutes of each meeting.

SUMMARY

Certain operational components of the Program Plan for 1975-76 have presented Staff with challenges beyond anticipation, others have been functional almost from inception and improving steadily, while a few components have been forced into a "no progress" state by factors beyond the control of anyone. All in all, it has been a productive year with a lot of ground covered and many problems resolved before they became real stumbling blocks.

Projections for 1976-77 fiscal year follow in Part III of this document. Realignment of program priorities will be the subject of important policy discussions at the Executive Committee and Advisory Council fall meetings.

III

PROGRAM PLAN 1976-77

Goals and Objectives

A. COMMUNICATIONS AND DATA SYSTEM

Goal 1. All women in Arizona who may have problems during the period surrounding childbirth are identified as to the degree of risk before the problems occur.

Objective. Physicians and nurses in at least 10 Arizona hospitals will be routinely using uniform clinical records which identify pregnant women on the basis of low, medium or high risk by July 31, 1977.

Proposed Activities.

1) Complete revision and second field test of the following clinical records by January 1, 1977: Prenatal, Hospital Admission, Labor-Delivery, and Discharge Summary.
2) Complete second field test of Neonatal I (Normal Newborn Nursery) and Neonatal II (Newborn Intensive Care Nursery) clinical records by March 1, 1977.
3) Revise clinical records based on results of second field test, print and distribute within two months of completion of second field tests.
4) Prepare an orientation program in use of uniform clinical records for presentation to physicians and nurses by January 1, 1977.
5) Conduct orientation programs in at least six locations in Arizona by April 1, 1977.
6) Periodically review the clinical records for completeness, ease of use, accuracy of terms and educational values in light of changing clinical technology.
7) Conduct orientation programs in four additional locations in Arizona by June 1, 1977.

Goal 2. Perinatal health professionals throughout Arizona have immediate access to consultation with obstetricians and neonatologists in Tertiary Care Centers in order to provide superior management of special problems which may occur to women during the period surrounding childbirth.

Objective. To have in operation an efficient and effective voice communications system between Tertiary Care Centers and referring physicians by November 1, 1976.

Proposed Activities.

1) Complete arrangements with Catalina Communication Systems for purchase and installation of mobile VHF radio telephone units for use by September 1, 1976.
2) Complete technical orientation on use of equipment for users by September 15, 1976.
3) Develop and conduct a comprehensive information

program which will reach all potential referring physicians and will explain procedures for obtaining immediate consultation with obstetricians and neonatologists in Tertiary Care Centers by November 1, 1976.

4) Develop a simple evaluation tool for use in a periodic review of the effectiveness of the system by November 1, 1976.

5) Conduct semi-annual evaluation from users of the system and make changes as necessary.

Goal 3. All perinatal events which might influence the management of pregnancy are uniformly collected and transferred to a central repository where information can be easily and immediately retrieved when needed for patient care, as well as stored for future use in clinical and program studies.

Objective. Perinatal health professionals in at least 10 hospitals will routinely be using uniform clinical records and transmitting data to the central computer at St. Joseph's Hospital by July 31, 1977.

Proposed Activities.

1) By September 1, 1976, develop recommendations for the flow of clinical records from their source to the point of computer data entry in order to assure prompt, efficient and consistent handling of the forms and data entry.

2) In collaboration with medical and administrative personnel in five selected hospitals, analyze patterns of staffing and specific individual conditions in order to provide the most effective method of adapting procedures for record flow by November 15, 1976. Select an additional five hospitals for discussion of procedures to be completed by February 15, 1977.
Recommend the installation of, and periodically review the effectiveness of such data communication links between installations as are necessary to facilitate computer data entry of clinical records.

3) At least every six months the Communications and Data System Committee will review the effectiveness of computer procedures and programs for entering and retrieving computerized data, and suggest new procedures or enhancements to existing procedures that would aid in the effective utilization of the computer system.

4) At least every six months the Communications and Data System Committee will review procedures of computer and data security so as to (a) insure the viability of the system in case of accident, and (b) insure the confidentiality of patient data in keeping with ethical and current legal considerations.

5) By October 1, 1976, develop guidelines for responding to external requests for patient data which will insure patient data confidentiality.

Goal 4. Families and referring physicians of transport patients have access to a toll-free telephone system with Tertiary Care Centers which facilitates communications with Center personnel concerning the care and well being of transport patients.

Objective a. By March 1, 1977 complete testing and evaluation of a prototype telephone system at Tucson Medical Center's high-risk new-born nurseries which makes possible out-going direct dialing from the Center to any referral hospital or physician in the State and allows toll-free incoming calls to the Center by interested parties from five communities in Southern Arizona.

Proposed Activities.

1) Begin a six-month trial and evaluation period of the prototype telephone system by September 1, 1976.
The system will consist of an outgoing direct-dial touch-tone "business" line located in the Tucson Medical Center Intensive Care Nursery area, and an incoming special reverse long distance of an "enterprise" line located in the Intensive Observation (intermediate care) Nursery.

2) Conduct an evaluation of the system as follows:
   a) records of all calls during the six-month study period will be kept to document usage of both the incoming and outgoing telephone lines;
   b) retrospective comparison will be made of calling frequency before and after installation of the toll-free system;
   c) comparison will be made of parental call frequency from families in communities where toll-free service is not available;
   d) numbers of telephone consultations and rapidity of decision to transport will be compared before and after installation of the new communication system, and between the study and control communities;
   e) a questionnaire will be developed to canvas the opinions of staff personnel and parents as regards the convenience of the new system and what the added service has meant to them;
   f) develop a system of correlation with neonatal followup program using a control group.

Objective b. By May 1, 1977, determine if prototype telephone system is applicable to other areas in Arizona.

Proposed Activities.

1) If telephone system is successful, explore feasibility of installing system in other communities and Tertiary Care Centers.

2) Investigate various sources of funding for statewide network of such a system.

B. MATERNAL TRANSPORT SYSTEM

Goal 1. All high-risk maternity patients have access to a coordinated and efficient transport system which provides the means of transfer to the appropriate level of care.

Objective a. Appropriate hospital personnel in 20 Arizona hospitals with obstetrical units will be informed on site about procedures for transporting high-risk maternity patients by June 1, 1977.

Proposed Activities.

1) Complete final draft of "Protocol for Consultation and Maternal Transports" by August 20, 1976.

2) Distribute the "Protocol" to all hospital administrators, chief OB nurses, and chiefs-of-staff by September 15, 1976 along with general information about the Arizona Perinatal Program.

3) In collaboration with personnel at Tertiary Care Centers, develop a continuing education program which specif-

ically addresses the problems and procedures of transporting high-risk maternity patients by November 15, 1976.

4) Based on available neonatal morbidity and mortality data by hospital, select 10 hospitals with greatest need and schedule initial presentations on transport procedures by December 1, 1976.
5) Select appropriate medical personnel to conduct presentations on transport procedures by December 1, 1976.
6) Complete initial presentations on transport procedures by March 1, 1977.
7) Schedule presentations in 10 additional hospitals by March 1, 1977.
8) Complete presentation by June 1, 1977.

Objective b. To conduct an ongoing evaluation of the efficiency and effectiveness of the maternal transport system and make changes as necessary.

Proposed Activities.

1) Continue collection and analysis of relevant data on maternal transports including cost information, time studies, number and origin of transports, patient data on both mother and infant, and problems encountered during transport.
2) Based on an analysis of data collected, modify transport system as needed.
3) Prepare evaluation report on transport procedures by July 1, 1977.

C. COORDINATION OF SOCIAL SERVICES

Goal 1. All high-risk maternity patients and families of patients have access to social workers' care in order to minimize the incidence of family crises related to transport and other operational Program components.

Objective. Clearly define the role and involvement of social work in the perinatal care system in Arizona by April 1, 1977.

Proposed Activities.

1) Collect available data to determine the extent and availability of social workers involved in perinatal care in Arizona by December 1, 1976.
2) Convene representatives from social work, medicine and nursing to discuss the comprehensive long-range development of a plan for including social work in the perinatal care system in Arizona by February 1, 1977.
3) Complete the plan by April 1, 1977.

D. EDUCATION

Comment: Target dates for educational activities are dependent upon Arizona Perinatal Program receiving additional funds to employ a Director of Education. As of July 14, 1976, informal notification of approval of a grant request to the Grand Canyon-Saguaro Chapter of the National Foundation March of Dimes has been received. When formal notification of final approval is received, recruitment for a Director of Education will be finalized.

A proposed education plan has been developed in outline and is included as Appendix #3 in the unabridged annual report.* Only major goals and objectives are presented here.

Goal 1. Appropriate continuing education and training programs in perinatal care are conducted through-out the state in order to assure that well-trained health professionals are available, both in terms of numbers and geographical distribution, to provide high-quality perinatal care to all high-risk mothers and infants in Arizona.

Objective a. By month X, first year, a statewide plan for providing and coordinating continuing education programs for perinatal health professionals will be completed. The plan will include recommendations for the type of programs to be conducted and locations in which programs should be initiated.

Objective b. Publish a bi-monthly newsletter which will be distributed to selected physicians, nurses, hospital administrators and other health professionals.

Goal 2. Patients, families of patients and the general public are well informed about high-risk pregnancies the importance of perinatal and related health care and the location of and means of access to perinatal services in Arizona.

Objective a. By month X, first year, a statewide plan for patient and family education, including recommendations for implementation will be completed.

Objective b. By month X, first year, a statewide plan for a public education program will be developed.

Goal 3. All medical and nursing students in Arizona are informed about and exposed to fundamental concepts of high-risk perinatal care in basic education programs.

Objective a. Determine the extent to which students in relevant health professions are exposed to these concepts currently.

Objective b. In collaboration with appropriate faculty members, develop plans to include these concepts in basic education courses for medical and nursing students.

E. GRADED CARE FACILITIES

Goal. Three categories or levels of care facilities (primary, secondary and tertiary care centers) are available in Arizona to provide obstetrical and neonatal care consonant with risk levels of patients.

Objective. To assure that at least 10 hospitals in Arizona are considered as Primary, Secondary and Tertiary Care Centers in accordance with the APP "Recommendations - Perinatal Care Centers" by July 31, 1977. This will entail specifying one additional Tertiary Care Center, four Secondary Care Centers and five Primary Care Centers.

Proposed Activities.

1) Complete the draft of "Recommendations - Perinatal Care Centers" and distribute copies to appropriate hospital staff and perinatal health professionals for review by August 15, 1976.
2) Begin collecting and analyzing data including 1975 neonatal and fetal death rates for each hospital in Arizona by October 1, 1976.

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*All Appendices are on file in the Arizona Perinatal Program Office, Phoenix, and copies are available upon request.

3) On the basis of data analysis and initial interest of hospital staff, select 10 hospitals for preliminary field trips to explore further interest of staff in becoming perinatal care centers by January 1, 1977.
4) In collaboration with personnel from potential care centers, determine manpower, education/training and facility requirements for participation for each potential center by February 1, 1977.
5) Develop plans for assisting potential centers to follow APP recommendations for becoming a participating perinatal care center in the regional perinatal care system in Arizona by March 1, 1977.
6) Develop standard procedures whereby institutions can become participating centers in the regional perinatal care system in Arizona by January 1, 1977.

F. LABORATORY SPECIMEN TRANSPORT SYSTEM

Goal. All perinatal health professionals have access to specialized diagnostic laboratories in Arizona for testing non-routine laboratory specimens.

Objective. Perinatal health professionals in at least 10 hospitals in Arizona will be routinely using the transport system for non-routine laboratory specimens by July 31, 1977.

Proposed Activities.

1) Complete a written protocol for specimen preparation and transport to Arizona Medical Center laboratory in Tucson and St. Joseph's Hospital, Department of Reproductive Medicine laboratory in Phoenix by November 1, 1976. The protocol will include plans for reporting to referring physicians no later than six hours after tests are completed.
2) Distribute protocol to all outlying hospitals by December 1, 1976.
3) Complete contracts with public carriers for transporting specimens by November 15, 1976.
4) Solicit comments and suggestions from users of the system and prepare a report by May 1, 1977.

G. FOLLOW-UP OF HIGH-RISK PATIENTS

Goal. All high-risk mothers and infants will have access to appropriate followup care.

Comments: Further development of followup plan for neonates will be deferred until Staff has reviewed uniform one-year followup protocols under development by the Johns Hopkins group. (See "Followup of High Risk Patients," pp 10 ff.)

The Newborn Follow-up Program, Department of Pediatrics, University of Arizona will continue the long term, neonatal followup program as described in Part II, pp 10 ff. It is our understanding that consideration will be given by the Department of Pediatrics to expand the scope of the study in order that it may be submitted as part of a comprehensive research proposal in the field of neurophysiologic development to another funding agency.

Maternal followup plans will be developed as indicated in Part II of this document.

H. ADMINISTRATION

Goal 1. A program plan which provides direction to the Arizona Perinatal Program (APP) will be revised annually. The plan will include a summary of activities during the previous year and goals and objectives for the current year.

Objective. The Second Annual Report will be completed by August 15, 1977.

Proposed Activities.

1) Prepare a summary of activities for each program component quarterly for Executive Committee and Advisory Council review and comment. Quarterly reports will be due November 15, February 15, May 15, and August 15.
2) Consolidate quarterly reports and prepare Annual Report by August 15, 1977.
3) Distribute Annual Report to the Robert Wood Johnson Foundation, Executive Committee, Advisory Council and other appropriate individuals and organizations by August 15, 1977.

Goal 2. The organization of the Arizona Perinatal Program is structured to allow maximum participation by health professionals and consumers in the planning, implementation and evaluation of the perinatal system in Arizona.

Objective a. The Advisory Council will meet at least biannually to recommend general program policies, goals and objectives and review program direction.

Proposed Activities.

Standing committees and special committees appointed by the President will meet as necessary. Minutes of meetings will be sent to all Advisory Council and Executive Committee members.

Objective b. The Executive Committee will meet at least bimonthly to review staff activities and recommendations, hear reports from the Advisory Council, ad hoc Task Groups and others, and make policy decisions.

Proposed Activities.

1) The Executive Committee will review the By-laws and Constitution and determine if revision is appropriate by November 1, 1976.
2) If necessary, By-laws and Constitution will be revised by December 1, 1976.

Goal 3. Resources required for the continuation and expansion of the perinatal care system in Arizona will be developed by 1980.

Objective. To develop financial and other resources which match anticipated Program growth.

Proposed Activities.

1) By November 1, 1976 determine priority of committed funds and the amount of financial resources which will be required for each Program component in August 1977.
2) Continue development of various resources for future payment of Program activities as well as possibilities for resource development for Program components currently inadequately funded.
3) Consider alternative methods for developing a comprehensive resource development plan such as outside consultant versus staff activity.

I. EVALUATION

Goal. The efficiency and effectiveness of the Arizona Perinatal Program as well as the impact on the health status of mothers and infants in Arizona will be ascertained through an evaluation process shared with the Health Services Research and Development Center, Johns Hopkins University.

Proposed Activities.
Comments: Baseline studies outlined in Part II, p. 15, will be continued, and vital data forwarded from the Arizona Department of Health Services' Vital Records Section, as outlined.

IV

ARIZONA PERINATAL PROGRAM

Cash Disbursements
Fiscal Year Ending July 31, 1976

Account	Cash Disbursements For Fiscal Year 1975-76
Administration	$ 129,345.04
Screening Instrument	3,875.10
Services	58,684.93
Transport	—0—
Communications & Data System	27,359.42
Education	4,007.52
Follow Up Care	8,748.10
Evaluation	—0—
Total	$ 232,020.11

Robert Wood Johnson Foundation Grant #1636 Arizona Medical Association Foundation, Inc.

Clinical data bases (data sheets) completed and presently ready for initial major field tests are contained in Appendix #1 of the unabridged annual report.

Data sheets will be batched in most Primary Care Centers and forwarded to APP headquarters for entry into the central computer. Secondary and Tertiary Care Centers will use input/retrieval terminals with a capability of instant response from the central computer. Both input and retrieval modes will be the "branching menu" tape with an appropriate number of options both on input and retrieval.

From the computer center at St. Joseph's Hospital and Medical Center in Phoenix, card image tapes will be forwarded to Doctor Shapiro's group in the format and sequence requested.

HIGHLIGHTS FROM THE STATE AUXILIARY

We are pleased to resume publishing some of the Auxiliary happenings. Their contributions to our professional image is considerable, and for the most part unsung.

—Editor

County Auxiliary Presidents reported plans for active programs throughout the state. Mini-review: **Coconino** ladies were the gracious hostesses for that County Society's summer (festival time) medical seminar, and wives attending were all raves over their attentions and special activities arranged for them. **Maricopa** plans another super Spree Market for Nov. 9th. **Mohave** Auxiliary members expect to participate in their local Health Fair, and **Pima** plans a Concerns for Women workshop among the many scheduled activities under their "Caring and Sharing" theme. **Yavapai's** 35 members have a full slate ahead including involvement with members-at-large in the Sedona area and the Flagstaff auxilians when they hostess a combined gathering honoring state officers. **Yuma** wives are busily raising funds for the new hospital.

State Board action included reducing the state newsletter "Caduceus Crier" to three issues this year in partial compensation of withdrawn Blue Shield support funds of recent years. The A.M.A. Education and Research Fund received almost 1½ million dollars from the Auxiliary last year. The new goal is two million: a thought to remember when asked to purchase a ticket on a hand-painted shotgun . . . or something.

William B. McGrath, M.D., has been appoined medical director of Camelback Hospital, effective November 1, 1976.

Dr. McGrath has been in private practice in psychiatry in Arizona since 1947 and is a fellow of the American Psychiatric Association. He was among the founders of the Arizona Foundation for Neurology and Psychiatry which was absorbed into Camelback Hospital in 1962. Since that time he has served continually on the board of trustees.

Author of two books, *Mental Fitness* (1967), and *The Heart Does Not Speak English* (1975), Dr. McGrath is widely known throughout the state of Arizona and has served on the board of directors of the Maricopa County Society and the board of the Arizona State Hospital. He has also served as chairman of the board of the Industrial Relations Committee and the medical advisory board of the Arizona State Industrial Commission.

In Memoriam

SO LONG, HARRY . . . .

HARRY T. SOUTHWORTH
1904 — 1976

Your leaving us was sudden though not surprising, because we knew you wished it so. Why then do tears well up in eyes looking at today when your contributions were yesterday? We'll try to tell you why.

One could start by saying that your life began in Prescott where it ended. Your Dad, who died while you were still at Rush, was a loved Prescott M.D., also, and duly recognized. We searched the paper for a similar reflection of love recognizing you and your contribution to your community. We finally found it . . . a few facts stacked among the other obituaries. Sound disappointed? You bet!

It was you we met time and again at 3 a.m. returning from the Old Ball Park area after tending to Rosita's G B Colic or some similar emergency. Thinking of the Old Ball Park, we are reminded that you were one heck of a short stop in your growing up days, wanted to coach and did some of same while in the Air Force in World War II. If you had taken the path of baseball, perhaps you would have had more recognition today.

Then there were the many OB's: the bleeders, the breaches in primipara, the pre-eclamptics. You were never too busy to lend a hand. How vividly we remember the near term OB brought to the hospital after an auto accident. Seconds after she died you had the uterus open trying to save her unborn child. We wonder how many namesake Harrys there are around because of a mother's devotion to her doctor.

Surgery? How you would anticipate old Earnie's every move; tying, cutting, retracting, making the proper remark at the right time to break tension and relieve frayed nerves. Earnie never openly showed his appreciation, but we knew there was a bond between you two that was never broken.

Charts? Boy, were you ever always and constantly behind! How we tried to get you to write PH, U ch D F Inq, essentially negative FH, no TBC, CA or Diabetis; but not you. Your charts were beautifully written, most in long hand: detailed, covering negative as well as positive findings—an internist's dream.

Politics? A handful of M.D.'s in the valley will remember that you were president of the Arizona Chapter of A.A.G.P. and of ArMA. The exact dates escape us, but you handled those jobs in your precise, detailed, efficient way. And what a great party fellow you were! That contageous laugh of yours still rings in the ears.

We could go on recollecting . . . about a lonely life after the untimely death of your wife, Margaret; how proud you were of son Tommy and daughter Kaki; of sister Dorothy Faye married to Tex Ritter . . . how your last days were spent in almost total blindness. But there is a point to be made.

The "so long" to you, Harry, is a good-bye not only to a special person, but to a fading phase of medicine. Though you drove a big car, you were a "Country Doc", and Country Docs are fast disappearing.

We've spent our tears, Harry. We are proud to have touched elbows with such a great guy.

Mell Phillips and the many doctors
who really knew you
(Prescott)

ArMA
Medical History

ST. JOSEPH'S HOSPITAL GROWS FROM COTTAGE TO MEDICAL CENTER

J. N. TANGDELIUS

One hot afternoon in the summer of 1892, Sisters of Mercy Mary Peter and Mary Alcocoque were walking along the dirt streets of Phoenix on an errand. They saw a young man sitting in a chair outside a rooming house, gasping for breath. They helped him to his bed, did what little they could for him, and promised to return with medicine the next day. And return they did—only to find that the young man had died during the night.

The Sisters had come to Phoenix with Mother Mary Paul in answer to the town's urgent cry for teachers. But they began to realize that the town needed a hospital even more than the school they had come to build. So they set out with horse and buggy, going from house to house and ranch to ranch, asking for funds to build a new hospital. By Feb. 21, 1895, the hospital was open.

It was only a small cottage with 6 rooms and 12 beds, and it was known to Phoenicians simply as "Sisters Hospital." Surgery was often done in patients' rooms, since there was no operating room. The operating table was a 7-foot pine board, 2-feet wide, placed on 2 sawhorses. Only 2 types of anesthetics were known—ether and chloroform. Ether was the favorite, and was poured directly from the bottle into a funnel and administered to the patient. (Four to six people were generally required to hold the patient on the board until the anesthetic took effect.) But despite this and other primitive techniques of the day, operations had a high rate of success.

The first St. Joseph's - 1895.

The second St. Joseph's in 1896. Fire Destroyed this building in 1917; it was rebuilt in 90 days.

More rooms were soon needed, and less than a year later the Sisters moved into a new building with 24 rooms. It was close to the town's irrigation ditch, which presented a few problems. One night a seriously ill patient became delirious and fled from the hospital, only to fall into the water of the ditch. The Sisters waded in and rescued their patient—but the patient had made a discovery. Later, he would threaten to jump in the ditch again unless he could have exactly what he wanted.

By 1901, the hospital had to be expanded again. An annex was built to provide space for more patient rooms, an operating room, a diet kitchen and a utility room. In 1907 a second operating room was added, and in 1910 the first school of nursing in Arizona was established.

By 1913, a nurses' home had been added, and the hospital consisted of 3 buildings. The main building included 50 private rooms, 2 large operating rooms, consulting rooms, a reception room and baths. The annex featured 22 "elegantly furnished suites." Departments included medical, surgical and maternity. Rates for the private rooms in the main building were $20 to $40 per week; rooms in the obstetrical department were

The Nursey in 1915 seems primitive in comparison with modern perinatal facilities.

This was modern equipment in the 1917 Maternity Department.

$30 per week, or with a private bath, $35 and $40 per week; and the wards, located in the main building, cost $10 to $14 per week.

In October, 1917, a disastrous fire destroyed the main hospital, but the annex and the nurses' home were saved, and there was no loss of life. Reporting on the fire, the *Arizona Republican* said: "It was remarkable the way help came though it seemed long until the fire department reached the scene and when it did it finally had to take water from the town ditch as the hydrant pressure proved insufficient for such a big fire." High school boys helped carry patients out of the buring building, and private cars were drafted to transport them to the annex, nurses' home and private homes so their care could be continued. Men worked in 3 shifts to rebuild the hospital, and in 90 days the building had been reconstructed on the same site.

The new hospital boasted 140 beds by 1922. Arrangements for the "making and keeping of case records" had been completed, and a new laboratory and x-ray had been installed. The new surgery was finished and equipped, and a new third story had been added to the nurses's home.

By 1947, the city's explosive growth had outdistanced the hospital's facilities once more, but so many departments, rooms and wings had been added that there was no longer room for growth in the existing hospital building. The Sisters of Mercy launched another drive for funds, this time for an entirely new St. Joseph's, to be built on land bought from the Central Avenue Dairy Co. Plans were made, funds donated by the community, the medical staff, and other contributors, and building began. In 1953 St. Joseph's moved again — to the hospital's present site on Thomas Road between 3rd

The Hospital in 1952. A year later, St. Joseph's moved to its present site on Thomas Road.

and 5th avenues.

Services have continued to expand, keeping pace with the demands of the growing community. Mercy Clinic was established in 1955 to provide medical services at a cost adjusted to each patient's ability to pay. 1956 marked the arrival of a kidney dialysis machine—the first in an Arizona hospital. In 1962, the Barrow neurological Institute was established. Two years later, the Mental Health Center—the first of its kind in a general Phoenix hospital—and the Coronary Care Center were opened. In 1967, the first Newborn Intensive Care Nursery was opened at St. Joseph's, a cryosurgical unit for ophthalmology and otolaryngology patients was installed, a Respiratory Intensive Care Unit was opened, and the first computer cardiogram in Arizona was made.

Services continued to develop. In 1969, the first genetic counseling service was made available, the Narcotic Addiction and Rehabilitation Program was begun, a spinal cord injury center was established, a gastroenterology laboratory was opened, the first hemophilia referral center in Arizona and a referral center for treatment of epilepsy were established, and a helistop, linked to a statewide system of medical evacuation by air, was constructed.

The 70's have seen further growth. The Arizona Heart Institute was opened in 1971, as was the Department of Reproductive Medicine. 1972 marked the opening of the Pediatric

Intensive Care Unit to provide a type of medical care not available in the more specialized intensive care units. A special sensory laboratory was established in 1973 in the Barrow Neurological Institute; and in that year lay members were named to the Hospital's board of directors—a new concept in the management of St. Joseph's

The Perinatal Laboratory was greatly expanded in 1974, and the following year St. Joseph's was named as a regional center for the Arizona Regional Perinatal Program, and as a recipient of a grant from the Robert Wood Johnson Foundation for the development of that program.

But the needs continue to grow, too. The Hospital recently made application for a certificate of need for new facilities to expand the present bed capacity of 560 to 779 and to provide expanded laboratories and other services. Oncology radiation facilities are expected to be completed by January, 1977.

St. Joseph's is actively engaged in medical research, particularly in functions involving the brain and spinal cord; and in education, training medical residents and in cooperation with local colleges and universities, nurses, technicians and technologists.

St. Joseph's is governed by the Sisters of Mercy of Burlingame, Calif., who also operate five hospitals in California. Sister M. Assumpta Murray is the president of St. Joseph's board of directors. Executive director and administrator is T. Abner Huff.

JOHN LAWRENCE LE CONTE, M.D.

PART II

REV. EDWARD J. PETTID

We conclude the adventures of Dr. Le Conte as he explores in the southwest.

Very sincerely yours
John L. LeConte

The Major's previous conversation with Dr. Ogden first sowed the dragon seeds of doubt about Dr. Le Conte; but after a secret conversation with Juan the Sonoranian the Major doubled his doubts about Le Conte.

This Juan the Sonoranian, Dr. Le Conte's guide, had in the course of a private conversation with Major Heintzelman stated that silver abounded in Sonora. In fact, it was so abundant that he, Juan, with his very own eyes had seen a house in Alamos raftered with solid silver.

This did it. This confirmed the strongest suspicion of the Major that Doctor Le Conte's strong interest in hay and beetles was merely a tricky camouflage for a stronger interest for solid silver.

Actually, the Major's suspicions about Doctor Le Conte were misdirected. The Major should have suspicioned himself. For, seven years later, on 10 July 1858, after an absence of four years from the West Coast and after becoming a Colonel, S. P. Heintzelman passed through San Diego with a pocket plan for Tubac, Arizona Territory: where he speculated in the Heintzelman Mines in the Altar Valley immediately north of the United States-Mexican borderline.

Major Heintzelman worte in his *Journal* several pet names the eminent Medical Doctor who preferred people to call him "John". The Major nicknamed him "Dr. Bugs" or the "bug doctor".

However, Dr. Le Conte and Juan, his Sonoranian guide, did leave Fort Yuma. While watching patiently for the Doctor's return with a pot silver, the Major questioned, on 9 and 19 February 1851, several migrating Mexican sheepherders about him but with negative results.

What had happened to the Doctor? cutting away from the Colorado River and riding eastward on the Gila River trail, Dr. Le Conte and Juan, mounted on horses saddled with leather borrowed without permission by Lt. Sweeny from government quartermaster supplies, traversed more than a hundred miles up the Gila River by Agua Caliente, Painted Rocks, and from the big bend of the Gila River shortcutted south of the Sierra Estrella mountains to Maricopa Wells near the old Pima Villages.

And all the while being the thorough scientist, like a hawk, he searched for black beetles in the tree-like saguaro cactus on the Arizona desert, along the Gila riverbanks below the Pima Villages, and beneath the Cottonwood tree bark in the valley of the Colorado and Gila Rivers. And it can now be reported: he searched

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From: Mercy Hospital & Medical Center, 4077 Fifth Ave., San Diego, CA 92103.

successfully and himself widely reported the success.

Shortly after arriving at Maricopa Wells, a middle-aged Dutch-American emigrant named Royse Oatman met Le Conte to inquire about the Fort Yuma road and to obtain a waybill.

This conversation was summarized and printed in the book, *Captivity Of The Oatman Girls*, authored by Royal Byron Stratton in 1857:

> "He had passed the whole distance to Fort Yuma, and returned, all within a few months, unharmed; and stated that he had not witnessed indications of even the neighborhood of Indians".

Lured further south by visions of new and more exciting species of beetles, Dr. Le Conte left Maricopa Wells for Picacho Peak and Pointer Mountain and Tucson.

Around 1 February 1851 while in Tucson, as the Doctor later reported:

> "I saw two large masses of iron, evidently meteorite, which were used as anvils by the two blacksmiths of that town. They were irregular in form, and although imbedded in the ground to make them steady enough for use, they were about three feet high. I endeavored to have some pieces cut off, and although a high price was offered, the characteristic Mexican indolence could not be overcome. The only answer I could obtain, was the metal was "muy duro".
>
> "These pieces were brought from a valley in a small mountain chain, about forty miles southeast of Tucson, east of the road leading to Tuvaca. In this valley, fragments similar to those seen, and of various sizes were said to be abundant. From the occurrence of this metal, the valley was called "Canada de Hierro", or iron valley. Silver mines of great richness are very numerous in that vicinity; the metal occurs as sulphuret, with galena and blende, and also in the native form".

Fully realizing there indeed existed an extreme forage and food shortage in the Pima Villages, Tucson and deeper South because of the great southwestern drought and constant Apache raids, the Doctor discarded his daydreams to profitably provide forage for Fort Yuma. Instead he concentrated on pure Science—and a side bonus—espionage.

While leisurely returning towards Fort Yuma, John came back to the Pima Villages. There the Coco-Maricopa Indians whispered into his ear that the Yuma Indians were treacherously inciting all the angry Indians along the Gila River emigrant trail to massacre the Whites. Quite troubled with this serious information the Doctor and Juan then hurried back down the Gila River to warn the Major and spread the alarm.

Retracing their waterless desert shortcut from Maricopa Wells, they loped the forty miles "not enough grass to pick a man's teeth" to the big bend of the Gila River by 15 February 1851 where they re-met the Oatman family sadly encamped on the Gila riverbank: Royse, the father, Mary Ann (Sperry), the very pregnant mother, and seven children ranging in age from sixteen to a one year old baby, bearing the names of Lucy, Lorenzo Dow, Olive Ann, Mary Ann, Royse, Jr., Charity Ann and Roland.

The Oatman family, a fraction of a Brewsterite party of disfellowshipped Mormons, had left Independence, Missouri on 5 August 1850 to form a new western colony. The Oatmans had separated themselves from their wagon train and were now alone and desperate. Their oxen, wagon, supplies and harness were in bad shape. On top of all, Mrs. Oatman daily expected a new baby.

In the *Captivity Of The Oatman Girls* this meeting between the Oatmans and Dr. Le Conte is described:

> ". . . were overtaken by Dr. Lecount, who with his Mexican guide was on his way back to Fort Yuma. The doctor saw our condition, and his large, generous heart poured upon us a flood of sympathy, which, with the words of good cheer he addressed us, was the only relief it was in his power to administer. Father sent by him, and at his own suggestion, to the fort for immediate assistance. This message the doctor promised should be conveyed to the fort, . . . with all possible dispatch, . . . We were all transiently elated . . . "

The next information on the Le Conte-Juan relief party emerged when a weary, dusty, footsore Juan stumbled into Fort Yuma on Friday 21 February with a four-pointed sad tale: first, he had left the Doctor back east about sixty miles on the Gila trail; next, he accused the Yuma Indians of stealing their horses; he advised the Major that the Yumas were arranging a rebellion; lastly, he delivered the letter from Royse Oatman begging for four horses to get him to the Colorado.

On Monday, 24 February, Dr. Le Conte finally arrived at Fort Yuma about 2:00 p.m. and made a fully formal visit to the Major's tent pitched in the original riverbottom camp just below the mouth of the slough among the arrow weed, willow, mesquite and cottonwood trees—at the old ferry, near Jaeger's. He peremptorily demanded a relief party be sent up the Gila River to some emigrants who needed horses—Royse Oatman and his family—about a hundred miles east.

Miffed with what he wrote was the Doctor's attitude of arrogance and presumption, the Major checked his short-fused temper while replying there were neither animals fit for any journey nor forage immediately available. Even the government wagon train could scarcely be outfitted with sufficient horses and mules to meet the supply ship INVINCIBLE now nosing into the mouth of the Colorado.

The Major reflected later about the report of the robbery of the animals from the Doctor and Juan and decided to speak with the Chiefs of the Yuma (Quechan) Indians, Santiago and Caballo y Pelo, about it. He also conversed with the Yumas about the rumors that they were conspiring with other Indians to murder the Whites. Unperturbed, the Indian Chiefs puffed the peace pipe and denied everything.

Meanwhile, in his Journal for Friday, 7 March 1851, the Major scribbled:

> "I must contrive some way to rid the camp of Dr. Bugs. I would think Dr. Hewit would be tired paying his meat bill. And I do not think his trip into Sonora was much of a speculation".

On Saturday, 8 March, the two soliders ordered to the relief of the Oatman family returned to Fort Yuma and reported the Oatman massacre. It was learned that there were three survivors: the boy Lorenzo and the two girls Olive Ann and Mary Ann. But the two girls were in Indian captivity.

Was Doctor Le Conte blameworthy for not staying with the Oatmans? Major Heintzelman thought he was:

> "Dr. Le Conte had so much charity he should have staid & accompanied the family, particularly as the woman expected daily to need his services. If he & his Sonoranian had staid & come on with them, they would probably not have lost their horses. Some people like charity at other peoples expenses".

Captain Davidson argued and pressed for a large relief expedition to rescue the girls from the Yavapai but trouble with Cocopah Indians downriver cancelled that relief expedition.

When Lorenzo Dow Oatman, the tomahawked son who had survived the massacre, arrived at Fort Yuma on Monday, 31 March 1851, Lt. Sweeny requested the use of the government boat for himself and then oarbladed Dr. Le Conte and head-injured boy to the Fort's hospital tents where Dr. Hewit was Surgeon.

It is difficult to decide whether Heintzelman misjudged Le Conte or no based on the following Heintzelman *Journal* entry:

> "Dr. Le Conte came up opposite here yesterday about 1 o'clock & fired his pistol & set fire to the shed the volunteers left; but no one heard him & he had to ride down & cross at the ferry. He did not reach here until some time after dinner. I wish him no more harm than that he had been on foot instead of a government mule. Mr. Sweeny should not have lent him. He had no business to burn those sheds. He has not been near me . . . I must contrive some way to drive Bugs off. This man cannot stay much longer".

By 3 April Heintzelman was no longer directly bugged by Dr. Le Conte. The Doctor was returning via San Francisco to New York. The Doctor swore that even $50,000 would not tempt him to remain on the Colorado River for another six months.

To review his financial interests and to prepare his paper, "An Attempt to Classify the Longicorn Coleoptera of the part of America North of Mexico", for reading before the Academy of Natural Science at Philadelphia on 16 September, the Doctor loaded down with tertiary fossils and many mounted beetle specimens sailed aboard the Pacific Mail's steamship, CALIFORNIA, on 15 June 1851 for New York.

From New York the Doctor moved his residence in 1861 to Philadelphia where he married Helen Grier and fathered two sons.

His future life became a rapid succession of scientific researches: in 1857 he officially accompanied the Honduras Inter-Oceanic Survey; in 1867 he was the geologist with General W. W. Wright on a survey for the Union Pacific Railway; from the Autumn of 1869 to the Winter of 1872 he scientifically surveyed and lectured in Europe, Algiers and Egypt; consultant and writer in Entomology for the Smithsonian Institution.

At the outbreak of the Civil War, Doctor Le conte volunteered for the Union Army medical corps as Surgeon of Volunteers, and advanced to the post of Medical Inspector with the rank of Lieutenant Colonel. In 1878 he was Director of the United States Mint at Philadelphia.

Doctor John Lawrence Le Conte died on 15 November 1883 at his 1625 Spruce Street residence in Philadelphia. About him his fellow doctors and scientists spoke and wrote words of the highest praise:

> "Le Conte was the greatest entomologist this country has yet produced is unquestionable . . . European entomologists who crossed the ocean paid him their first visit . . . . his wonderfully retentive memory, his quick and accurate judgment, his courage and self-reliance, all gave his words weight, and his counsel and opinion were eagerly sought by his conferers".

Dr. Le Conte justified this respect when he reflected his own well-balanced soul:

> "Let not our vanity lead us to believe that because God has deigned to guide our steps a few paces on the road to truth, we are justified in speaking as if He had taken us into intimate companionship, and informed us of all His counsels".

Dr. Le Conte echoed the Ages past and projected into the Ages to come: Science and Religion, complementary truths, should work "harmoniously for the perfection of man and the glory of his Creator".

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On principle it is high time to abandon the policy of licensure or board certification in perpetuity, not only because of anxiety over the pressures from the government and other outside sources, but also because some type of periodic review is essential to safeguard the public.

Bulletin, American College Surgeons, Sept. 1974, Vol. 59 #9.

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

THE ARIZONA MEDICAL ASSOCIATION, INC.

FINANCE COMMITTEE

The meeting of the Finance Committee of the Arizona Medical Association Inc. held at 810 West Bethany Home Road, Phoenix, Az on Saturday, August 28, 1976 convened at 1:44 p.m., John T. Clymer, M.D., Treasurer and Chairman, presiding.

MINUTES

The minutes of the meeting held March 6, 1976 were approved as distributed.

ArMA FINANCIAL STATEMENT — 7/31/76

Mr. Robinson and Mr. Griffin reviewed ArMA's financial statement for the first seven month period ending 7/31/76 explaining each account and how it is used.

CONDENSED STATEMENT OF REVENUES AND EXPENDITURES

For seventh month ending 7/31/76

	Actual	Under/(Over) Budget	Total Annual Budget
GENERAL DEPARTMENT			
Revenue	$423,484.15	$(20,934.15)	$402,550.00
Expenditures	244,846.63	99,553.37	344,400.00
	178,637.52	(120,487.52)	58,150.00
COMMITTEE DEPARTMENT			
Revenue	46,816.19	(26,816.19)	20,000.00
Expenditures	129,927.98	(72,127.98)	57,800.00
	(83,111.79)	45,311.79	(37,800.00)
ANNUAL MEETING			
Revenue	54,602.00	(602.00)	54,000.00
Expenditures	50,975.49	2,424.51	53,400.00
	3,626.51	(3,026.51)	600.00
PUBLISHING DEPARTMENT			
Revenue	36,240.20	16,059.80	52,300.00
Expenditures	43,302.19	26,397.81	69,700.00
	(7,061.99)	(10,338.01)	(17,400.00)
SUB-TOTAL OF GENERAL OPERATIONS			
Revenue	$561,142.54	$(32,292.54)	$528,850.00
Expenditures	469,052.29	56,247.71	525,300.00
	$ 92,090.25	$(88,540.25)	$ 3,550.00
BUILDING OPERATIONS			
Revenue	34,850.63	(17,100.63)	17,750.00
Expenditures	27,805.33	12,194.67	40,000.00
	7,045.30	(29,295.30)	(22,250.00)
BENEVOLENT & LOAN OPERATIONS			
Revenue	364.04	4,135.96	4,500.00
Expenditures	—0—	2,000.00	2,000.00
	364.04	2,135.96	2,500.00
TOTAL ALL OPERATIONS			
Revenue	$596,357.21	$(45,257.21)	$551,100.00
Expenditures	496,857.62	70,442.38	567,300.00
	$ 99,499.59	$(115,699.59)	$(16,200.00)

GENERAL DEPARTMENT

For the month of July and year-to-date 7/31/76

		ACTUAL		BUDGET	
		Month of JULY	Year-to-Date 7/31/76	Year-to-Date Under/(Over) Budget	Total Annual Budget
REVENUE					
401	AMA Commissions	$ 213.00	$ 4,573.70	$ (2,173.70)	$ 2,400.00
403	Dues—Active Members	1,105.00	363,320.00	(320.00)	363,000.00
404	Dues—Interns Residents	—0—	624.00	(74.00)	550.00
406	Dues—Service Members	—0—	662.00	38.00	700.00
421	Sale of Supplies	66.50	627.05	272.95	900.00
429	Other	939.38	7,536.00	(2,036.00)	5,500.00
420	Printing Dept. Income	4,097.08	46,141.40	(16,641.40)	29,500.00
	Total Revenue	6,420.96	423,484.15	(20,934.15)	402,550.00

EXPENDITURES				
501 Auditing	25.00	3,886.00	(886.00)	3,000.00
504 Dues, subs., cont.	89.00	2,396.00	(396.00)	2,000 00
505 Equipment repair & maint.	2,791.21	10,178.74	12,821.26	23,000.00
507 General O H (Recovery)	(333.33)	(2,333.31)	(466.69)	(2,800.00)
508 Insurance, general	6,423.38	13,745.38	7,254.62	21,000.00
509 Insurance, health	501.70	3,958.20	1,541.80	5,500.00
510 Legal	400.00	2,800.00	1,200.00	4,000.00
511 Meeting	7,169.25	14,326.63	4,673.37	19,000.00
512 Miscellaneous	223.74	885.18	1,614.82	2,500.00
513 Payroll taxes	480.79	6,632.95	3,167.05	9.800.00
514 Postage	660.01	3,194.69	5,805.31	9,000.00
515 Printing	565.60	2,569.13	730.87	3,300.00
516 Refund AMA Commissions	8,207.43	8,207.43	(3,207.43)	5,000.00
518 Salaries	15,187.92	105,538.74	85,461.26	191,000.00
519 Supplies	69.12	9,398.75	12,201.25	21,600.00
520 Telephone	796.57	6,955.23	3,044.77	10,000.00
521 Travel	1,949.51	6,742.54	5,757.46	12,500.00
522 President's Expense	1,018.59	2,378.23	2,621.77	5,000.00
525 Printing Dept. Expense	4,880.60	43,386.12	(43,386.12)	—0—
Tot Expenditures	51,106.09	244,864.63	99,553.37	344,400.00
	$(44,685.13)	$178,637.52	$(120,487.52)	$ 58,150.00

COMMITTEE DEPARTMENT

For the month of July and Year-To-Date 7 31 76

	ACTUAL		BUDGI	
	Month of JULY	Year-to-Date 7 31 76	Year-to-Date Under/(Over) Budget	al Annual Budget
REVENUE				
431 CME Processing Fees	$ 5.00	$ 125.00	$ (125.00)	$ —0—
432-4 MICC Assess. & Contrib.	3,650.00	9,590.00	(9,590.00)	—0—
433 MICA Commissions	2,486.52	37,101.19	(17,101.19)	20,000.00
	6,141.52	46,816.19	(26,816.19)	20,000.00
EXPENDITURES				
500A Articles of Inc. Bylaws	—0—	6.28	93.72	100.00
500C Data Coll. & Analysis	—0—	—0—	—0—	—0—
500D Board of Directors	1,087.98	2,745.81	54.19	2,800.00
500E Executive	11.67	1,024.81	(624.81)	400.00
500F Finance	—0—	76.62	23.38	100.00
500H Grievance	6.83	58.83	241.17	300.00
500I History & Obituaries	—0—	—0—	—0—	—0—
500J Maternal Child Hlth Care	—0—	176.75	2,023.25	2,500.00
500K Health Manpower	55.00	1,620.12	(620.12)	1,000.00
500L Legislative	892.79	7,025.06	2,174.94	9,200.00
500M Medical Education	83.74	1,276.12	3,723.88	5,000.00
500N Medical Economics	—0—	—0—	450.00	450.00
500O Occupational Health	—0—	—0—	350.00	350.00
500Q Professional	6.15	751.97	648.03	1,400.00
500R Public Relations	738.18	13,917.34	12,082.66	26,000.00
500S Government Services	—0—	245.45	4,954.55	5,200.00
500U Ad Hoc — House Staff	—0—	698.42	301.58	1,000.00
500W Woman's Auxiliary	—0—	1,200.00	—0—	1,200.00
500X Physicians Rehabilitation	—0—	186.00	(186.00)	—0—
500Y Professional Liab. Review	3,795.13	15,612.36	(14,812.36)	800.00
500Z MICC	474.26	83,006.04	(83,006.04)	—0—
Total Expenditures	7,151.73	129,927.98	(72,127.98)	57,800.00
	$ (1,010.21)	$(83,111.79)	$ 45,311.79	$(37,800.00)

ANNUAL MEETING REVENUE AND EXPENDITURES

For month of July and Year-to-Date 7/31/76

	ACTUAL		BUDGET	
	Month of JULY	Year-to-Date 7/31/76	Year-to-Date Under/(Over) Budget	Total Annual Budget
REVENUE				
441 Exhibit booth rental	$ —0—	$14,500.00	$ (400.00)	$14,100.00
442 Sponsorship, speakers	—0—	2,800.00	(800.00)	2,000.00
443 Steak Fry	—0—	5,489.00	711.00	6,200.00
444 President's Banquet	—0—	3,877.00	123.00	4,000.00
445 Breakfast Panel #1	—0—	736.25	13.75	750.00
446 Breakfast Panel #2	—0—	769.75	(19.75)	750.00
447 Registration Fees	—0—	18,430.00	(230.00)	18,200.00
448 B/S Share of Expense	—0—	8,000.00	—0—	8,000.00
449 Other	—0—	—0—	—0—	—0—
Total Revenue	—0—	54,602.00	(602.00)	54,000.00
EXPENDITURES				
541 Exhibit booth expense	—0—	1,477.60	1,522.40	3,000.00
542 Guest speaker expense	—0—	7,114.62	(614.62)	6,500.00
543 Steak Fry	—0—	7,436.45	3,763.55	11,200.00
544 President's Banquet	—0—	7,215.30	3,484.70	10,700.00
545 Breakfast Panel #1	—0—	726.00	(76.00)	650.00
546 Breakfast Panel #2	—0—	924.00	(274.00)	650.00
547 Audio-visual equipment	—0—	2,703.17	(103.17)	2,600.00
548 Delegate's Manual	—0—	4,542.87	(2,642.87)	1,900.00
549 Employee hotel	—0—	2,426.97	(26.97)	2,400.00
550 Gratuities, hotel emp.	—0—	225 00	25.00	250.00
551 Miscellaneous	—0—	2,966.90	(766.90)	2,200.00
552 Newcomer Program	5.00	40.00	45.00	80.00
553 Nominating Committee	—0—	36.26	63.74	100.00
554 Postage	—0—	—0—	400.00	400.00
555 Printing	—0—	3,954.10	(454.10)	3,500.00
556 Promotion	—0—	3,979.31	(979.31)	3,000.00
557 Public Relations	—0—	2,118.63	(618.63)	1,500.00
558 Sci. Assembly Comte.	—0—	1,116.74	(466.74)	650.00
559 Supplies	—0—	1,351.74	248.26	1,600.00
560 Travel	—0—	28.80	71.20	100.00
561 Telephone	—0—	591.03	(171.03)	420.00
Total Expenditures	5.00	50,975.49	2,424.51	53,400.00
	$ (5.00)	$ 3,626.51	$(3,026.51)	$ 600.00

PUBLISHING COMMITTEE REVENUE AND EXPENDITURES

For the month of July and Year-to-Date 7/31/76

	ACTUAL		BUDGET	
	Month of JULY	Year-to-Date 7/31/76	Year-to-Date Under/(Over) Budget	Total Annual Budget
REVENUE				
461 Advertising	$ 2,292.30	$12,796.63	$ 14,203.37	$ 27,000.00
462 Reprints	607.25	813.40	986.60	1,800.00
463 Subscriptions, member	65.00	22,330.00	(330.00)	22,000.00
464 Subscriptions, nonmember	20.00	249.60	1,250.40	1,500.00
465 Other	—0—	50.57	(50.57)	—0—
Total Revenue	2,984.55	36,240.20	16,059.80	52.300.00

EXPENDITURES				
504 Dues and subscriptions	60.00	120.00	80.00	200.00
505 Equipment maintenance	—0—	—0—	50.00	50.00
506 General overhead	333.33	2,333.31	2,066.69	1,100.00
508 Insurance, general	—0—	224.58	325.42	550.00
509 Insurance, health	19.50	156.00	144.00	300.00
511 Meeting	—0—	87.50	112.50	200.00
512 Miscellaneous	—0—	—0—	—0—	—0—
513 Payroll taxes	26.58	342.58	257.42	600.00
514 Postage	—0—	600.00	300.00	900.00
515 Printing	4,725.25	33,668.67	18,331.33	52,000.00
518 Salaries	400.00	5,152.50	4,747.50	9,900.00
519 Supplies	27.80	559.55	(559.55)	—0—
521 Travel	—0—	57.50	542.50	600.00
Total Expenditures	5,592.46	43,302.19	26,397.81	69,700.00
	$(2,607.91)	$(7,061.99)	$(10,338.01)	$(17,400.00)

BUILDING OPERATIONS
For the month of July and Year-to-Date 7/31/76

	ACTUAL		BUDGET	
	Month of JULY	Year-To-Date 7/31/76	Year-to-Date Under (Over) Budget	Total Annual Budget
REVENUE				
471-3 B/F Assessments	$4,725.00	$25,925.00	$(14,775.00)	$ 11,150.00
474 BOMEX rental	691.75	4,842.25	1,757.75	6,600.00
475 ARPP	583.34	4,083.38	(4,083.38)	—0—
Total Revenue	6,000.09	34,850.63	(17,100.63)	17,750.00
EXPENDITURES				
571 Depreciation expense	1,100.00	7,700.00	4,300.00	12,000.00
572 Repair & maintenance	234.14	3,590.59	1,409.41	5,000.00
574 Building supplies	45.05	1,312.23	(412.23)	900.00
575 Interest expense	—0—	165.63	3,834.37	4,000.00
576 Property taxes	400.00	6,767.90	(1,767.90)	5,000.00
577 Utilities	1,810.51	9,528.98	3,971.02	13,500.00
578 Insurance	—0—	—0—	2,000.00	2,000.00
579 Bldg. O/H (Recovery)	(180.00)	(1,260.00)	(1,140.00)	(2,400.00)
Total Expenditures	3,409.70	27,805.33	12,194.67	40,000.00
	$2,590.39	$ 7,045.30	$(29,295.30)	$(22,250.00)

BENEVOLENT AND LOAN OPERATIONS
For the month of July and Year-to-Date 7/31/76

	ACTUAL		BUDGET	
	Month of JULY	Year-to-Date 7/31/76	Year-to-Date Under (Over) Budget	Total Annual Budget
REVENUE				
481 Interest Income	$ —0—	$ 364.04	$4,135.96	$4,500.00
EXPENDITURES				
504 Scholarships Awarded	—0—	—0—	2,000.00	2,000.00
	$ —0—	$ 364.04	$2,135.96	$2,500.00

BALANCE SHEET

July 31, 1976

ASSETS

	Current assets		
101	Petty cash fund	$ 50.00	
102	Cash in bank, general account 66-6582	4,206.74	
103	Cash in bank, petty cash account 66-6566	1,000.00	
110	Expense Advance	—0—	
	Accounts Receivable		
109	Special Malpractice Assessment Receivable	7,000.00	
111	Advertising and miscellaneous	10,969.95	
112	Building Fund, due in one year	10,600.00	
114-5	Inventories, food service and printing supplies	5,838.31	$ 39,665.00
	Long-term investments and funds		
108	Note Receivable MICA	519,588.28	
112	Building Fund, due after one year	48,942.50	
145	Cash surrender value of life insurance	34,980.91	603,511.69
	Fixed assets		
152	Building, net of accumulated depreciation	358,447.60	
153	Furniture and fixtures, net of accumulated depreciation	35,762.79	394,210.39
	Other assets		
160	Organization costs	265.75	
170	Guarantee deposits	4,068.20	
180	MICA	60,622.89	
190	Transfer of funds	(5,043.90)	
191	Food Service clearing account	1,566.52	
194	Practice Management Workshop	(97.25)	61,382.21
	TOTAL ASSETS		$1,098,769.29

LIABILITIES & FUND BALANCES

	Current liabilities		
201	Notes Payable Unsecured	$150,000.00	
202	Accounts payable	16,788.65	
203-4	AMA Dues unremitted	3,160.00	
217-18	Payroll taxes withheld & Savings bond	529.30	
231	CME Application Fees unremitted	10.00	
260	Retirement Agreement, due in one year	6,000.00	$176,487.95
	Other liability		
208	Note Payable — VNB	519,588.28	
260	Retirement Agreement, due after one year	14,500.00	534,088.28
	Fund Balances		
301	General Operations, January 1, 1976	58,173.03	
	Excess of Revenue over Expenditures, current period	92,090.25	
371	Building Operation, January 1, 1976	213,023.16	
	Excess of Expenditures over Revenue, current period	7,045.30	
381	Benevolent & Loan Operations	17,497.28	
	Excess of Revenue over Expenditures, current period	364.04	388,193.06
	TOTAL LIABILITIES AND FUND BALANCES		$1,098,769.29

MONTHLY LIST OF CHECKS

Mr. Robinson reviewed the Board of Director's action on 6 12 76 when it directed that members of the Finance Committee receive a list of all checks issued each month. He pointed out that should any member have a question about any check, they are to call him and he will provide an explanation.

ARIZONA MEDICAL ASSOCIATION FOUNDATION ARIZONA PERINATAL PROGRAM

Mr. Robinson reported that the Robert Wood Johnson Foundation Grant has just completed its first year of operation and the following is a report of that activity. (see pages 10 through 18 inclusive)

PROJECT APACHE

The following is the financial statement for the first month's operation of Project Apache which was taken over by the Arizona Perinatal Program on 7 1 76.

STATE OF
REVENUE AND
EXPENDITURES

FOR THE MONTH OF JULY
AND YEAR-TO-DATE: 7 31 76
FISCAL YEAR ENDS: 6 30 77

	ACTUAL		BUDGET	
	Month of JULY	Year-to-date 7 31 76	Year-to-Date Under (Over) Budget	Total Annual Budget
Revenue:				
401 DHS Grant	$37,666.68	$37,666.68	$188,333.32	$226,000.00
Total Revenue	37,666.68	37,666.68	188,333.32	226,000.00
Expenditures:				
A Personal Services	14,187.00	14,187.00	177,191.00	191,378.00
B Prof. & Outside Services	320.00	320.00	7,120.00	7,440.00
C Travel	1,089.50	1,089.50	12,730.50	13,820.00
D Occupancy Expense	(259.50)	(259.50)	3,571.50	3,312.00
E Other Operating Expense	90.53	90.53	7,609.47	7,700.00
F Equipment	439.95	439.95	1,910.05	2,350.00
Total Expenditures	$15,867.48	$15,867.48	$210,132.52	$226,000.00

ROBERT WOOD JOHNSON FOUNDATION GRANT #1636
RECAP

Original Budget Code	Revised Budget Code	Account	Detail on Page Number	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
1	500	Administration	2	$127,860.00	$129,345.04	$ (1,485.04)
2	600	Screening Instrument	3	19,500.00	3,875.10	15,624.90
3	700	Services	4	167,000.00	58,684.93	108,315.07
4	800	Transport	5	19,000.00	—0—	19,000.00
5 & 8	900	Communications & Data Systems	6	62,000.00*	27,359.42	34,640.58
6	1000	Education	7	19,250.00	4,007.52	15,242.48
7	1100	Follow Up Care	8	25,000.00**	8,748.10	16,251.90
9	1300	Evaluation	9	—0—	—0—	—0—
		TOTAL BUDGET		$439,610.00	$232,020.11	$207,589.89

*Original budget was $37,000.00, see note on "Communications & Data Systems" page 6, for explanation.

**Original budget was $40,000.00, see note on "Communications & Data Systems" page 6, for explanation.

ADMINISTRATION — OBJECTIVE #1

Original Budget Code	Revised Budget Code	Account	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
A	501	Executive Director Salary	$ 52,000.00	$ 52,100.08	(100.08)
B	502	Administrative Assistant Salary	15,000.00	11,350.00	3,650.00
C	503	Secretary	8,500.00	8,092.00	408.00
D	506	Employee Benefits	13,230.00	10,701.42	2,528.58
—	508	Payroll Taxes	—0—	3,491.20	(3,491.20)
E	510	Travel	8,100.00	3,156.57	4,943.43
F	520	Legal and Audit	4,575.00	4,146.12	428.88
G	525	Rent	7,000.00	7,000.08	(.08)
—	527	Telephone	—0—	3,250.59	(3,250.59)
H	530	Data Processing	4,000.00	—0—	4,000.00
I	540	Supplies and Expenses	4,800.00	4,124.96	675.04
J	555	Office Equipment	5,000.00	12,388.68	(7,388.68)
K	535	Printing	4,000.00	3,831.81	168.19
L	550	Accounting Service	1,655.00	1,849.00	(194.00)
	560	Meeting Expense	—0—	2,521.97	(2,521.97)
	565	Periodicals and Publications	—0—	442.62	(442.62)
	570	Equipment Maintenance and Rental	—0—	259.80	(259.80)
	575	Postage	—0—	638.14	(638.14)
		TOTAL ADMINISTRATION	$127,860.00	$129,345.04	$(1,485.04)

Budget Objective #1 is to formally organize the program by establishing an Executive Committee and appointing a full-time Executive Director with supporting staff and facilities. The line item approach is self explanatory and has been used for this budget objective only. Funds realized by investment will be placed in a special account for use as designated by the Executive Director.

SCREENING INSTRUMENT — OBJECTIVE #2

Original Budget Code	Revised Budget Code	Account	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
A	610A	Research and develop instrument to screen and identify high risk mother and infants	$ 1,500.00	$ —0—	$ 1,500.00
B	610B	Field test & evaluate instrument	3,000.00	—0—	3,000.00
C		Implement through appointment of staff person to educate health professionals in region to use instrument	15,000.00	3,875.10	11,124.90
		TOTAL SCREENING INSTRUMENT	$19,500.00	$3,875.10	$15,624.90

Budget Objective #2 has been estimated from projection of personnel type and time necessary to develop this portion of the overall perinatal system from data provided by the Arizona Division of Health Services and the University of Arizona, Tucson. Fiscal support for many of the phased screening objectives has already been accomplished through matching funds.

SERVICES — OBJECTIVE #3

Original Budget Code	Revised Budget Code	Account	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
A	710A	Designate Perinatal Program Directors	$ 24,000.00	$ 7,284.93	$ 16,715.07
B	711B	In-hospital High Risk Pregnancy	138,000.00	51,400.00	86,600.00
C	716C	Est. regional transportation system for lab. samples	5,000.00	—0—	5,000.00
		TOTAL SERVICES	$167,000.00	$58,684.93	$108,315.07

Budget Objective #3 has been estimated from existing cost data obtained from University Hospital, Tucson; St. Joseph's Hospital and Good Samaritan Hospital, Phoenix; and from the Arizona Newborn Transport and Intensive Care Program. With regard to 3B, it is estimated that 25% of the cost related to delivery of high risk pregnancy care will be reimbursed by third party payors. Funds applied against the residual costs incurred by families and institutions as a liability have been estimated at approximately $1200.00 per in hospital high risk pregnancy evaluation. It is equivalent to $69,000.00 per bed, per year at 100% utilization. The medical impact and fiscal efficiency of this budgetary approach to facilitating establishment, definition and program utilization has been established in the Arizona Newborn Transport and Intensive Care Program.

TRANSPORT — OBJECTIVE #4

Original Budget Code	Revised Budget Code	Account	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
A	810A	Train medical and para medical personnel for transport teams	$ 1,000.00	$ —0—	$ 1,000.00
B	816B	Integrate maternal transport into existing newborn transport system	18,000.00	—0—	18,000.00
		TOTAL TRANSPORT	$19,000.00	$ —0—	$19,000.00

Budget Objective #4 relates to the known cost of initially identifying and training designated perinatal transport teams in specific locations utilizing existing training programs. Other aspects of such training programs pertaining to continuing education and development of new transport teams in regionally defined locations will be accomplished under budget item 6. Eighteen thousand dollars has been estimated to be required to underwrite the costs of maternal transport when such transport cannot be directly related to fetal or newborn transport for which funds are already available from the Arizona Newborn Transport and Intensive Care Program.

COMMUNICATIONS AND DATA SYSTEMS* — OBJECTIVES 5 AND 8

Original Budget Code	Revised Budget Code	Account	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
A	900A	Establish intra-regional tele-communications system	$12,000.00	$ 81.69	$11,918.31
B	900B	Develop & implement a maternal fetal neonatal data transmission system	50,000.00*	27,277.73	22,722.27
		TOTAL COMMUNICATIONS AND DATA SYSTEMS	$62,000.00	$27,359.42	$34,640.58

**Original budget figure was $25,000.00. John M. Thoen's letter of 2/4/76 approved consolidating budget categories "Communications" and "Data Systems". ($10,000.00) The same letter authorized transferring $15,000.00 from the "Follow-Up Care" account to this account bringing this item to $50,000.00.*

Budget Objective #5 has been calculated directly from known costs for the installation of intra-regional telephone communication systems which will allow use of these mechanisms for patient care consultation, education and date transmission.

Budget Objective #8. The cost for coordination and development of the data system has been estimated from the known cost of these areas of activity at Arizona State University and the University of Arizona in relation to already active programs (dominately Project Apache and the Arizona Newborn Transport and Intensive Care Program). Coordination of the biomedical and computer engineering education functions of the universities with the information system available through the Arizona Health Planning Authority and with the Arizona Perinatal Program will result in both reduction of undue cost to the program for data acquisition and evaluation and increased efficiency of the data system.

EDUCATION — OBJECTIVE #6

Original Budget Code	Revised Budget Code	Account	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
A	1000A	On site visits to referred hospitals	$ —0—	$ 99.30	$ (99.30)
B	1000B	Physician & nursing continuing education programs	3,500.00	997.50	2,502.50
C	1000C	Dial-A-Tape program development	750.00	—0—	750.00
D	1000D	Tuition & Fee support for formal education programs for perinatal allied health professionals	10,000.00	—0—	10,000.00
E	1000E	Development of patient and consumer education programs	5,000.00	2,910.71	2,089.28
		TOTAL EDUCATION	$19,250.00	$4,007.52	$15,242.48

Budget Objective #6 has been calculated from data developed at the University of Arizona, pertaining to personnel requirements for expansion and maintenance of new and continuing educational services related to perinatal care.

FOLLOW UP CARE — OBJECTIVE #7

Original Budget Code	Revised Budget Code	Account	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
A	1110A	Follow up care program for 200 patients at an estimated cost of $200 pt	$25,000.00*	$8,748.10	$16,251.90
		TOTAL FOLLOW UP CARE	$25,000.00	$8,748.10	$16,251.90

**Original budget was $40,000.00. $15,000.00 transferred to "Communications and Data Systems" — see note on page 6.*

Budget Objective #7 has been estimated by assuming a cost of $200 per patient to initially evaluate the neurological status of 200 patients in two defined locations. The $40,000.00 thus estimated to be required to accomplish this program goal assumes that personnel and institutions responsible for these functions will also be involved in educational and screening activities of the program and may derive additional support from program activities in these areas.

EVALUATION — OBJECTIVE #9

Original Budget Code	Revised Budget Code	Account	Original Grant Budget	Actual Expenditures For Year	Carry Over Funds
A	1300A	Evaluation of data for quarterly performance reports	$ —0—	$ —0—	$ —0—
		TOTAL EVALUATION	$ —0—	$ —0—	$ —0—

Budget Objective #9. This item was originally estimated at $2,000.00 directly from estimates of personnel time required to evaluate certain components of the data system as they relate specifically to program impact evaluation. The item was reduced to $0 because of budget restrictions. It is our plan to fund this activity either from the Administrative budget or from other matching funds not presently identified.

BALLANCE SHEET
JULY 31, 1976

ASSETS

100 Petty Cash	100.00	
102 Cash in Bank #64-9803	22,023.88	
Total Assets		$22,123.88

LIABILITIES AND FUND BALANCES

LIABILITIES

225 State Income Tax Withheld	324.68	
Total Liabilities		324.68

FUND BALANCES

321 General Operations, July 1, 1976	—0—	
Excess of Revenue over expeditures, current period	21,799.20	21,799.20
Total Liabilities & Fund Balances		$22,123.88

ZERO BASED BUDGETING

Mr. Robinson reported on the Board of Director's action of May 1, 1976 directing that the "Zero Based" budgeting concept be used in preparing the 1978 budget for the Association.

It was understood that each committee chairman would be asked to review all activities of his committee with an eye to evaluating the effectiveness of existing programs as well as seriously looking at ways to reduce expenditures during the calendar year 1977 and to reduce the 1978 budget when it is preapred.

It was determined that the other two divisions of the budget; i.e. General Department and Building Operations, would be assigned to sub-committees of the Finance Committee who would review them and make recommendations to the full committee.

It was also recommended that when it comes time to finalize the 1978 budget, that at least two meetings be held so that ample time would be available for full discussion of all items. Announcements of those meetings to be as far in advance of the meeting date as possible.

OTHER BUSINESS

Financial Reports

It was determined to provide members of the committee with copies of the monthly financial statements of all three fiscal programs ie ArMa, ArMA Foundation's Project Apache and Arizona Perinatal Program.

Meeting adjourned 3:46 p.m.

William E. Crisp, M.D.
Secretary
by
Bruce E. Robinson
Executive Director.

DRUG ABUSE TREATMENT UNIT
(A New Narcotic Treatment Program)

Operated by the Institute for Human Services, Inc.

Ronald F. Hilding, M.D., Medical Director of the Program
Robert H. Barnes, M.D. Henri A. Moyal, M.D. Francis A. Enos, Ph.D.
Richard E. McKenzie, Ph.D. Andrew F. Miller, Ph.D.

Services now available.

The Unit will provide specific care for individuals addicted to narcotics, either street drugs or major pain relieving drugs. It will involve three treatment modalities: methadone maintenance, methadone detoxification, and drug-free. Counseling services include the following areas: individual, family, marital, group, biofeedback, hypnosis.

Methadone dispensing will occur only at the Phoenix office, located in the St. Luke's Medical Building, 421 North 18th Street, Suite 103, Phoenix. (602) 257-8089.

Counseling will be scheduled at the above office as well as our office at Los Arcos Medical Plaza, 1402 North Miller Road, Suite 9, Scottsdale.

The Unit is meant to provide a narcotic treatment program for those persons able to pay or reluctant to participate in programs labeled as providing care exclusively for drug addiction. The weekly fee includes daily methadone when necessary, random weekly urine drug screen, and one hour of psychotherapy.

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

THE FOLLOWING INSTITUTIONS HAVE RECEIVED ArMA ACCREDITATION FOR CONTINUING MEDICAL EDUCATION.

ARIZONA STATE HOSPITAL, PHOENIX
DESERT SAMARITAN HOSPITAL, MESA
GOOD SAMARITAN HOSPITAL, PHOENIX
PHOENIX INDIAN MEDICAL CENTER
MARICOPA COUNTY GENERAL HOSPITAL, PHOENIX
MEMORIAL HOSPITAL, PHOENIX
ST. LUKE'S HOSPITAL AND MEDICAL CENTER, PHOENIX
ST. JOSEPH'S HOSPITAL AND MEDICAL CENTER, PHOENIX
TUCSON HOSPITALS MEDICAL EDUCATION PROGRAM, TUCSON
VETERANS ADMINISTRATION CENTER, PRESCOTT

CONTINUING MEDICAL EDUCATION ACTIVITIES SPONSORED BY THESE INSTITUTIONS RECEIVE CATEGORY 1 CREDIT FOR THE ArMA CERTIFICATE IN CONTINUING MEDICAL EDUCATION AND THE AMA PHYSICIAN'S RECOGNITION AWARD

DERMATOLOGY CLINICAL CONFERENCE

November 29, 1976 — 5:15 p.m.
Tucson Medical Center, Tucson, AZ

SPONSOR: U of A College of Medicine
Dept. of Internal Medicine,
Dermatology Section

CONTACT:
Peter Lynch, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

HYPERTENSION WORKSHOP

November 17, 1976
Mesa Lutheran Hospital
Mesa, AZ

SPONSOR: Mesa Lutheran Hospital — MEDCOME

CONTACT:
J. Lloyd, M.D.
443 N. Alma School
Mesa, AZ

Approved for 4 required hours toward the ArMA Certificate in Continuing Medical Education.

24TH ANNUAL MEETING OF THE MEDICAL SOCIETY OF THE UNITED STATES AND MEXICO

November 10-13, 1976
Puerto Vallarta, Mexico
Holiday Inn

SPONSOR: Medical Society of the United
States and Mexico

CONTACT:
Mrs. Virginia E. Bryant
Executive Secretary
333 W. Thomas Rd., Ste 207
Phoenix, AZ 58013

BASIC SCIENCE REVIEW SEMINARS

November 16, 1976
December 21, 1976
7-9:30 p.m., Arizona Medical Center, Rm. 3505

SPONSOR: U of A College of Medicine

CONTACT:
George D. Comerci, M.D.
Office of Continuing Medical
Education & Outreach
U of A College of Medicine
Tucson, AZ 85724

THIRD INTERNATIONAL COCCIDIOIDOMYCOSIS SYMPOSIUM

November 17-19, 1976
Tucson, AZ

SPONSOR: American College of Chest
Physicians, U of A, Arizona
Regional Medical Program

CONTACT:
American College of Chest Physicians
911 Busse Highway
Park Ridge, Illinois 60068

MONTHLY CLINICAL SEMINAR (RECENT ADVANCES IN DIAGNOSIS & THERAPEUTICS)

November 20, 1976
Doubletree Inn, Tucson, AZ

SPONSOR: Dept. of Family & Community
Medicine, U of A, College of
Medicine

CONTACT:
Anthony F. Vuturo, M.D.
Dept. of Family & Community
Medicine, U of A
College of Medicine
Tucson, AZ 85724

Approved for 12 required hours toward the ArMA Certificate in Medical Education.

NATIONAL CONFERENCE ON SHORT TERM INTENSIVE PSYCHIATRIC HOSPITAL TREATMENT

November 20-21
Scottsdale Hilton Hotel, Scottsdale, AZ

SPONSOR: Camelback Hospital & National
Association of Private Psychiatric
Hospitals.

CONTACT:
Stuart M. Gould, Jr., M.D.
Camelback Hospital
5055 N. 34th Street
Phoenix, AZ 85018

Approved for 12 required hours toward the ArMA Certificate in Continuing Medical Education.

AMERICAN ASSOCIATION FOR CLINICAL IMMUNOLOGY AND ALLERGY

November 28-December 2, 1976
Braniff Place Hotel, Tucson, AZ

SPONSOR: American Association for
Clinical Immunology & Allergy

CONTACT:
Howard Silber, Exec. Dir.
American Assoc. for Clinical
Immunology & Allergy
104 S. 39th St.
Box 912
Omaha, Nebraska 68101

HYPERTENSION WORKSHOP

November 22, 1976
St. Lukes Hospital, Phoenix, AZ

SPONSOR: St. Hospital (Cardiac
Conference) — MEDCOME

CONTACT:
H. Hull, M.D.
525 N. 18th St.
Phoenix, AZ

Approved for 4 required hours toward the ArMA Certificate in Continuing Medical Education.

BIRTH DEFECTS & GENETIC COUNSELING IN PEDIATRIC PRACTICE

November 27-28, 1976
Univ. Hospital Auditorium, Tucson, AZ

SPONSOR: Arizona Chapter, American Academy of Pediatrics

CONTACT:
Thomas R. Harris, M.D.
Dept. of Pediatrics
College of Medicine
Tucson, AZ 85721

Approved for 7 1/4 required hours toward the ArMA Certificate in Continuing Medical Education.

GASTROINTESTINAL DIAGNOSIS

December 4 & 5, 1976
Maricopa County General Hospital
Phoenix, AZ

SPONSOR: Department of Radiology

CONTACT:
Department of Radiology
Maricopa County General Hospital
2601 East Roosevelt
Phoenix, AZ 85008

Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

HYPERTENSION WORKSHOP

December 13, 1976
John C. Lincoln Hospital, Phoenix, AZ

SPONSOR: John C. Lincoln Hospital — MEDCOM

CONTACT:
Eugene Ross, M.D.
444 W. osborn
Phoenix, AZ 85013

Approved for 4 required hours toward the ArMA Certificate in Continuing Medical Education.

STAFF QUARTERLY MEETING

December 15, 1976
Chimney's Restaurant
Glendale, AZ

SPONSOR: Glendale Samaritan Hospital

CONTACT:
Gerald Marshall, M.D.
6010 W. Northern
Glendale, AZ 85301

Approved for 1 required hour toward the ArMA Certificate in Continuing Medical Education.

FOURTH ANNUAL ARIZONA SURGICAL SYMPOSIUM

January 16-21, 1977
Doubletree Inn, Scottsdale, AZ

SPONSOR: Phoenix Surgical Society

CONTACT:
H. W. Kimball, M.D.
333 West Thomas Road
Phoenix, AZ 85013

Approved for 25 required hours toward the ArMA Certificate in Continuing Medical Education.

CURRENT CONCEPTS IN ANTIBIOTICS

January 29-30, 1977
Scottsdale Hilton, Scottsdale, AZ

SPONSOR: Health Maintenance Associates

CONTACT:
A. D. Jacobson, M.D.
2141 E. Camelback Rd.
Phoenix, AZ

Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

FILM READING SESSIONS & SCIENTIFIC MEETINGS

Monthly

SPONSOR: Phoenix Radiology Society

CONTACT:
Mrs. Mary Wood
810 W. Bethany Home Rd.
Phoenix, AZ 85013

Approved for 2 required hours per session toward the ArMA Certificate in Continuing Medical Education.

CLINICAL IMMUNOLOGY, ALLERGY AND RHEUMATOLOGY ROUNDS

Every Friday Noon-1 p.m.

Every Friday Noon-1 p.m.

SPONSOR: U of A College of Medicine
Dept. of Internal Medicine,
Clinical Immunology Section

CONTACT:
John Boyer, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

ENDOCTRINOLOGY SEMINAR

Every Thursday, Noon-1 p.m.
1st, 3rd, & 5th Thursday — Rm. N318, VA Hospital
2nd & 4th Thursday, Rm. 6505,
Tucson Medical Center

SPONSOR: U of A College of Medicine
Department of Internal Medicine

CONTACT:
Charles Nugent, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

HEMATOLOGY-ONCOLOGY CLINICAL CONFERENCE

Every Tuesday, Noon-1 p.m.
1st, 3rd, & 5th Tuesdays — Rm. 6505,
AZ Medical Center
2nd & 4th Tuesdays — Rm. N318,
Veterans Adm. Hospital

SPONSOR: U of A College of Medicine, Dept. of Internal Medicine

CONTACT:
Sidney Salmon, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

GRAND WARD ROUNDS — TRAUMA

Every Tuesday, 8 a.m.
Arizona Medical Center, Tucson, AZ

SPONSOR: U of A College of Medicine,
Surgery Dept., Trauma Section

CONTACT:
Martin Silverstein, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

MEDICAL GRAND ROUNDS

Every Wednesday, Noon-1 p.m.
1st, 3rd, & 5th Wednesday — Staff Conf. Rm., VA Hospital
2nd & 4th Wednesday — Rm 5403, Arizona Medical Center

SPONSOR: U of A College of Medicine
Dept. of Internal Medicine

CONTACT:
Jay Smith, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PROBLEM CASE WORKSHOPS

3rd Monday of each month 7:30 a.m.
Room 4410, Arizona Medical Center, Tucson, AZ

SPONSOR: Division of Ophthalmology, U of A
College of Medicine

CONTACT:
H. E. Cross, M.D., Ph.D.
Arizona Medical Center
Dept. of Surgery
Tucson, AZ

Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

PSYCHIATRIC GRAND ROUNDS

Every Wed., Sept. to May, 4-5:30 p.m.
Rm. 8403, Arizona Medical Center, Tucson, AZ

SPONSOR: U of A College of Medicine
Dept. of Psychiatry

CONTACT:
Alan Levenson, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 11/2 required hour per session toward the ArMA Certificate in Continuing Medical Education.

TRAUMA CONFERENCE

Every Monday, 4 p.m.
Rm. 4410, Arizona Medical Center, Tucson, AZ

SPONSOR: U of A College of Medicine
Dept. of Surgery, Trauma Section

CONTACT:
Martin Silverstein, M.D.
U of A College of Medicine
Tucson, AZ 85721

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

STAFF EDUCATION CONFERENCE

Wednesdays, Weekly, 1 p.m.
Arizona State Hospital, Phoenix, AZ

SPONSOR: Arizona State Hospital

CONTACT:
Howard E. Wulsin, M.D.
Arizona State Hospital
2500 E. Van Buren
Phoenix, AZ 85008

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

SURGICAL GRAND ROUNDS
4TH TUESDAY OF EACH MONTH

Hospital Auditorium
Baptist Hospital, Phoenix

SPONSOR: Baptist Hospital Phoenix

CONTACT:
James B. Shields, M.D.
6036 N. 19th Ave.
Phoenix, AZ 85015

Approved for 1½ required hours per month toward the ArMA Certificate in Continuing Medical Education.

PATIENT STAFFING CONFERENCE

Three times weekly
Camelback Hospital, Phoenix, AZ

SPONSOR: Camelback Hospital

CONTACT:
Stuart M. Gould, Jr., M.D.
Medical Director
Camelback Hospital
5055 N. 34th St.
Phoenix, AZ 85018

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CAMELBACK HOSPITAL CLINICAL CONFERENCE

3rd Tuesday monthly
Camelback Hospital, Phoenix, AZ

SPONSOR: Camelback Hospital

CONTACT:
Stuart M. Gould, Jr., M.D.
Medical Director
Camelback Hospital
5055 N. 34th St.
Phoenix, AZ 85018

Approved for 1 elective hour per session toward the ArMA Certificate in Continuing Medical Education.

COUNTER TRANSFERENCE GROUP

Weekly, Thurs. 8-10 p.m.

SPONSOR: Phoenix Psychiatric Council

CONTACT:
James E. Campbell, M.D.
5051 N. 34th St.
Phoenix, AZ 85018

Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

DESERT SAMARITAN HOSPITAL

Wednesday Evenings 7 p.m.

November 16 — Management of Carcinoma of the Breast. Tue. Evening—Fiesta Inn
December 1 Neonatal Meningitis

SPONSOR: Desert Samaritan Hospital

CONTACT:
L. A. Rosati, M.D.
Laboratory
Desert Samaritan Hospital
Mesa, AZ 85202

Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

GLENDALE SAMARITAN HOSPITAL MEDICAL EDUCATION LECTURES

January through December, 1976, 7 a.m.

SPONSOR: Glendale Samaritan Hospital

CONTACT:
Gerald Marshall, M.D.
6010 W. Northern
Glendale, AZ 85301

Approved for 5 required hours toward the ArMA Certificate in Continuing Medical Education.

PULMONARY DISEASE GRAND ROUNDS

Mondays – 12 Noon
D-5 North Conference Rm., Good Samaritan Hospital, Phoenix, AZ

SPONSOR: Pulmonary Disease Teaching Service
Good Samaritan Hospital

CONTACT:
Bernard E. Levine, M.D.
Pulmonary Function Laboratory
Good Samaritan Hospital
1033 E. McDowell Hospital
Phoenix, AZ 85006

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

CLINICAL CANCER CONFERENCE

3rd Wednesday Every Month
Butler Bldg. Conference Room
Good Samaritan Hospital, Phoenix, AZ

SPONSOR: Good Samaritan Hospital

CONTACT:
John A. Bruner, M.D.
926 E. McDowell Road
Phoenix, AZ 85006

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

BI-MONTHLY MEDICAL EDUCATION SEMINAR

Every other Wed. AM
Begin 7/3/74
Maryvale Samaritan Hospital, Phoenix, AZ

SPONSOR: Medical Staff Maryvale Hospital

CONTACT:
Thomas J. Groves, M.D.
6037 W. Elm St.
Phoenix, AZ 85033

Approved for 1 required hour per session toward the ArMA Certificate in Continuiing Medical Education.

TUMOR BOARD CONFERENCE

Every Friday at Noon
Kiva Conference Room
Phoenix Memorial Hospital

SPONSOR: Phoenix Memorial Hospital

CONTACT:
Howard Kimball, M.D.
333 West Thomas Road
Phoenix, AZ 85013

Approved for credit toward the ArMA Certificate in Continuing Medical Education.

MONTHLY MEDICAL EDUCATION SEMINAR

Third Monday of the Month
Kiva Conference Room
Phoenix Memorial Hospital

SPONSOR: Medical Staff of Memorial Hospital

CONTACT:
George Scharf, M.D.
1201 South 7th Avenue
Phoenix, AZ 85007

Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

MONTHLY MEETING OF TUCSON RADIOLOGISTS

Last Tues. of Month
Plaza International, Tucson, AZ

SPONSOR: U of A Medical Center, Dept. of Radiology

CONTACT: Irwin M. Freundlich, M.D.
Arizona Medical Center
Dept. of Radiology
Tucson, AZ 85724

Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

FAMILY PRACTICE CONFERENCE

1st Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Dir. of Medical Education
7300 E. 4th St.
Scottsdale, AZ

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

MORBIDITY & MORTALITY CONFERENCE

2nd Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Dir. Medical Education
7300 E. 4th St.
Scottsdale, AZ

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CLINICAL PATHOLOGICAL CONFERENCE

4th Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Director of Medical Education
7300 E. 4th St.
Scottsdale, AZ

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

MEDICAL GRAND ROUNDS

3rd Monday, Monthly, 12:45 p.m.
Scottsdale Memorial Hospital, Scottsdale, AZ

SPONSOR: Medical Staff

CONTACT:
R. C. Good, M.D.
Dir. of Medical Education
7300 E. 4th St.
Scottsdale, AZ

Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CARDIOLOGY CONFERENCE

Weekly —Friday 8-9 a.m.
St. Mary's Hospital Auditorium
Tucson, AZ

SPONSOR: St. Mary's Hospital

CONTACT:
A. L. Forte, M.D.
St. Mary's Hospital
Tucson, AZ 85724

Approved for one required hour toward the ArMA Certificate in Continuing Medical Education.

GRAND ROUNDS

Each Thursday 7 a.m.-8 a.m.
St. Mary's Hospital and Health Center,

SPONSOR: Depts. of Medicine, Surgery, Radiology, Pathology and Family Practice.

CONTACT:
Richard Silver, M.D.
Chairman, Medical Education
and Library Committee
Century Medical Plaza, Suite 160
1701 West St. Mary's Road
Tucson, AZ 85703

Approved for 1 required hour per round toward the ArMA Certificate in Continuing Medical Education.

ARIZONA MEDICINE

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CONTRIBUTIONS

The Editor sincerely solicits contributions of scientific articles for publication ARIZONA MEDICINE. All such contributions are greatly appreciated. All will be given equal consideration.

Material submitted for publication in ARIZONA MEDICINE should conform to the following policies:

1. Manuscripts, including references or bibliography, should be typewritten, double-spaced, on one side of the paper only, and the original and a carbon enclosed.

2. Be guided by the general rules of medical writing as followed by the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.

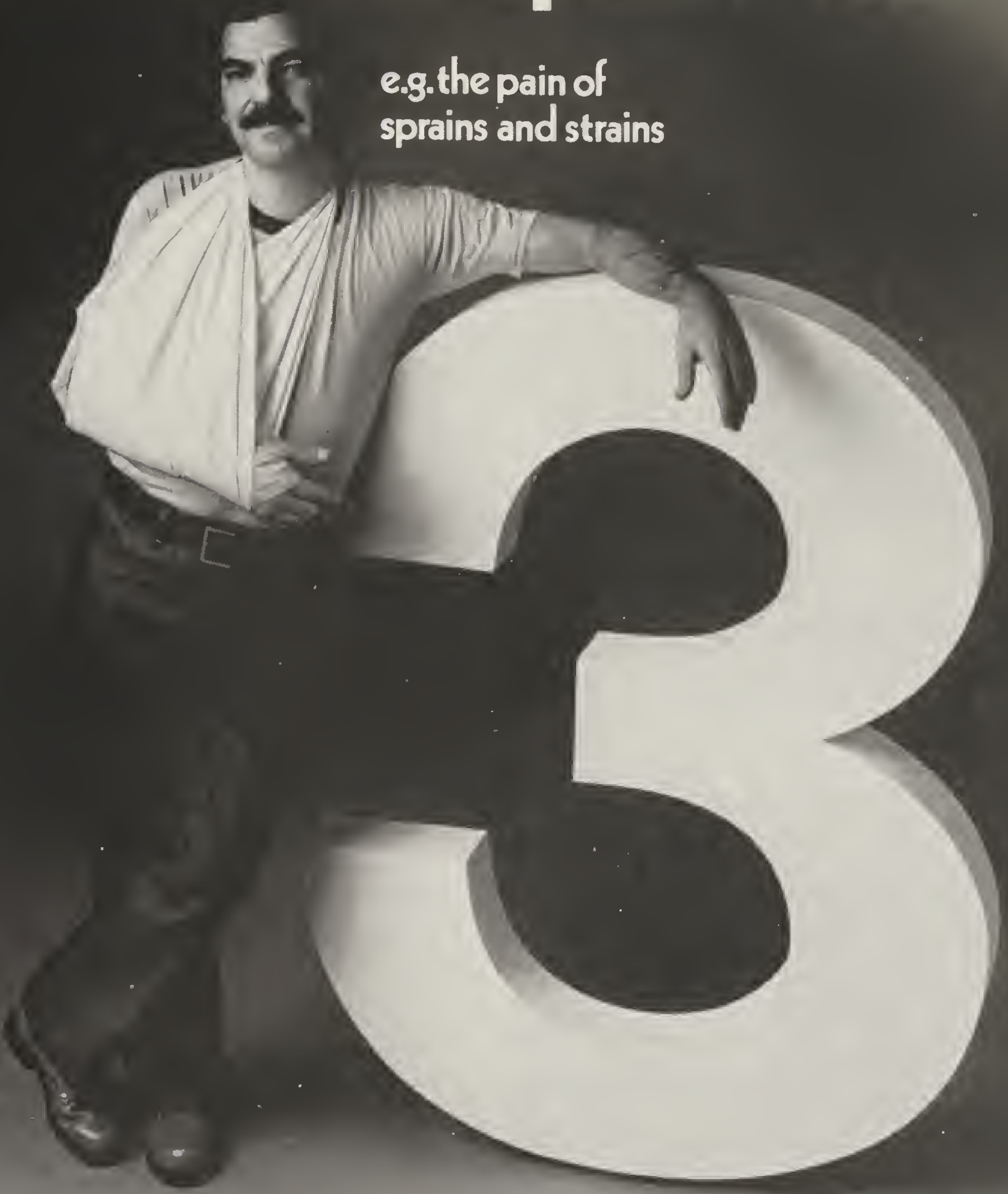
3. Although the Editors try to catch inaccuracies, the ultimate responsibility is the author's.

4. Articles are accepted for publication only if they are contributed exclusively to this Journal. Ordinarily, contributors will be notified within 60 days if a manuscript is accepted for publication. Every effort will be made to return unused manuscripts.

5. The Journal reserves the right to edit all material.

6. Reprints will be supplied to the author at printing cost.

No. 3
As potent as the pain it relieves.



e.g. the pain of
sprains and strains

OT TOO LITTLE

s potent as the pain you need to relieve in patients
vith fractures, sprains, strains, wounds, contusions,
nd the pain of surgical convalescence
nlike acetaminophen/codeine combinations, it
oes not sacrifice anti-inflammatory action

OT TOO MUCH

otent—yet not excessive ■ addiction liability low

NOT TOO EXPENSIVE

- brand-name quality, yet reasonable in cost
- readily available in both hospital and local pharmacies

Ⓒ CONVENIENCE

- telephone Rx in most states, up to 5 refills in 6 months at your discretion (where state law permits)

**MPIRIN® COMPOUND
ITH CODEINE NO. 3**

eine phosphate* (32.4 mg) gr ½
h tablet also contains: aspirin gr 3½, phenacetin gr 2½, caffeine gr ½. *Warning–may be habit-forming

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Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

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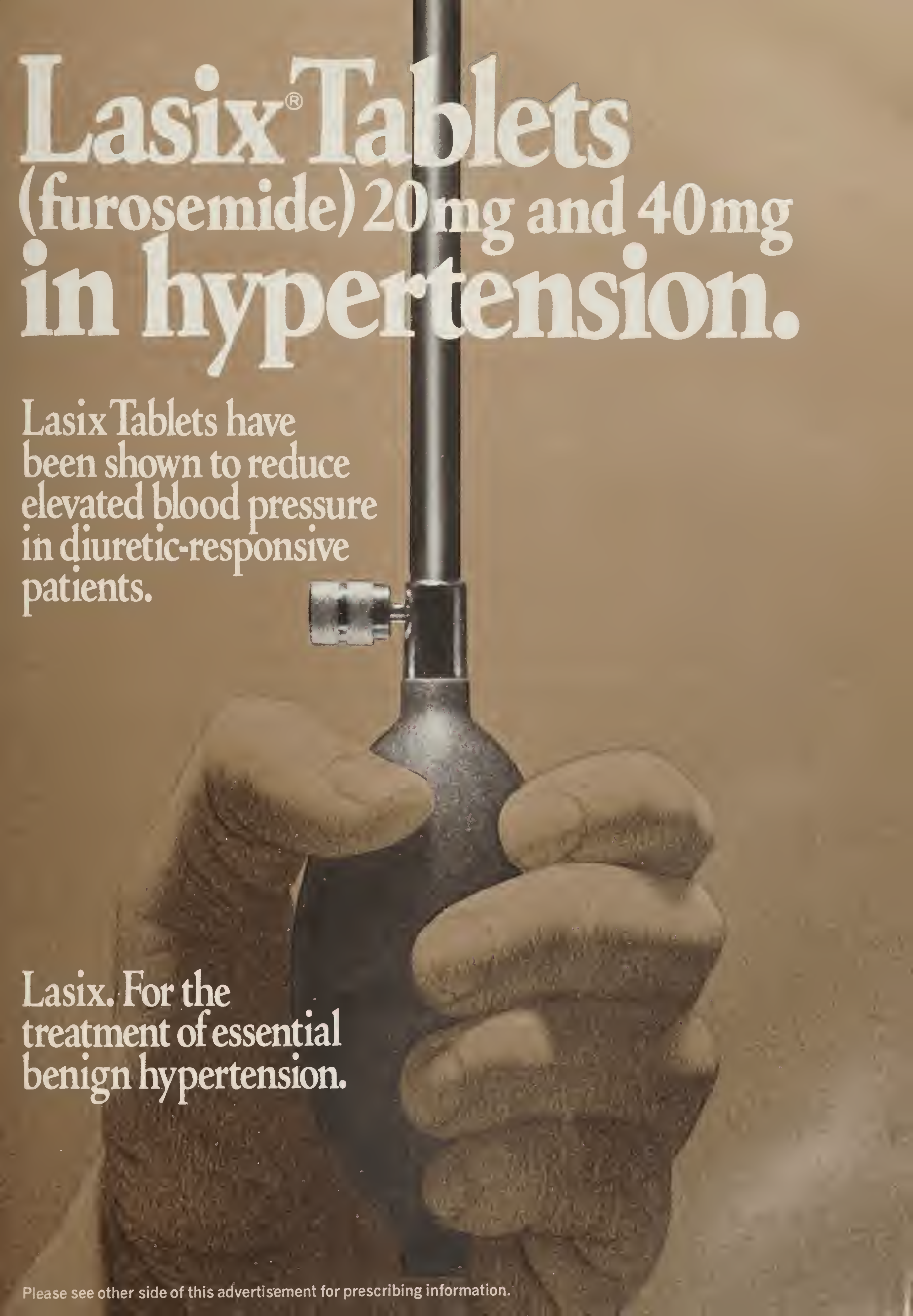

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Lasix® Tablets (furosemide) 20 mg and 40 mg in hypertension.

A brief summary of the Prescribing Information for

Lasix® (furosemide) Tablets 20 mg and 40 mg

WARNING—Lasix (furosemide) is a potent diuretic which if given in excessive amounts can lead to a profound diuresis with water and electrolyte depletion. Therefore, careful medical supervision is required, and dose and dose schedule have to be adjusted to the individual patient's needs. (See under "Dosage and Administration.")

Indications—Lasix (furosemide) is indicated for the treatment of the edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Hypertension—Lasix (furosemide) may be used for the treatment of hypertension alone or in combination with other antihypertensive drugs. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controllable with Lasix (furosemide) alone.

CONTRAINDICATIONS—Because animal reproductive studies have shown that Lasix (furosemide) may cause fetal abnormalities, the drug is contraindicated in women of childbearing potential. (See "Additional Information.")

Lasix (furosemide) is contraindicated in anuria. If increasing azotemia and oliguria occur during treatment of severe progressive renal disease, the drug should be discontinued. In hepatic coma and in states of electrolyte depletion, therapy should not be instituted until the basic condition is improved or corrected. Lasix (furosemide) is contraindicated in patients with a history of hypersensitivity to this compound.

Warnings—Excessive diuresis may result in dehydration and reduction in blood volume, with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients. Excessive loss of potassium in patients receiving digitalis glycosides may precipitate digitalis toxicity. Care should also be exercised in patients receiving potassium depleting steroids.

Frequent serum electrolyte, CO_2 and BUN determinations should be performed during the first few months of therapy and periodically thereafter, and abnormalities corrected or the drug temporarily withdrawn.

In patients with hepatic cirrhosis and ascites, initiation of therapy with Lasix (furosemide) is best carried out in the hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma; therefore, strict observation is necessary during the period of diuresis. Supplemental potassium chloride and, if required, an aldosterone antagonist are helpful in preventing hypokalemia and metabolic alkalosis.

Patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

In those instances where potassium supplementation is required, an oral liquid preparation should be used rather than enteric-coated potassium salts.

There have been several reports, published and unpublished, concerning nonspecific small-bowel lesions consisting of stenosis, with or without ulceration, associated with the administration of enteric-coated thiazides with potassium salts. These lesions may occur with enteric-coated potassium tablets alone or when they are used with nonenteric-coated thiazides, or certain other oral diuretics.

These small-bowel lesions have caused obstruction, hemorrhage, and perforation. Surgery was frequently required, and deaths have occurred.

Available information tends to implicate enteric-coated potassium salts, although lesions of this type also occur spontaneously. Therefore, coated potassium-containing formulations should be administered only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Patients with known sulfonamide sensitivity may show allergic reactions to Lasix (furosemide).

Precautions—As with any potent diuretic, electrolyte depletion may occur during therapy with Lasix (furosemide), especially in patients receiving higher doses and a restricted salt intake. Electrolyte depletion may manifest itself by weakness, dizziness, lethargy, leg cramps, anorexia, vomiting, and/or mental confusion.

Asymptomatic hyperuricemia can occur and gout may rarely be precipitated. Reversible elevations of BUN may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

When parenteral use of Lasix (furosemide) precedes its oral use, it should be kept in mind that cases of tinnitus and reversible hearing impairment have been reported. There have also been some reports of cases in which irreversible hearing impairment occurred. Usually, ototoxicity has been reported when Lasix (furosemide) was injected rapidly in patients with severe impairment of renal function at doses exceeding several times the usual recommended dose and in whom other drugs known to be ototoxic were often given. If the physician elects to use high dose parenteral therapy in patients with severely impaired renal function, controlled intravenous infusion is advisable (for adults, it has been reported that an infusion rate not exceeding 4 mg Lasix [furosemide] per minute has been used).

Increases in blood glucose, and alterations in glucose tolerance tests with abnormalities of the fasting and two-hour postprandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Lasix (furosemide) may lower serum calcium levels, and rare cases of tetany have been reported.

Patients receiving high doses of salicylates, in conjunction with Lasix (furosemide) may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Diuretics such as furosemide may enhance the nephrotoxicity of cephaloridine. Therefore, Lasix (furosemide) and cephaloridine should not be administered simultaneously.

Sulfonamide diuretics have been reported to decrease arterial responsiveness to pressor amines and to enhance the effect of tubocurarine. Great caution should be exercised in administering curare or its derivatives to patients undergoing therapy with Lasix (furosemide), and it is advisable to discontinue Lasix (furosemide) for one week prior to any elective surgery.

Adverse Reactions—Various forms of dermatitis, including urticaria and rare forms of exfoliative dermatitis, erythema multiforme, pruritus, paresthesia, blurring of vision, postural hypotension, nausea, vomiting, or diarrhea.

Anemia, leukopenia, aplastic anemia, and thrombocytopenia (with purpura). Rare cases of agranulocytosis which responded to treatment.

In addition, the following rare adverse reactions have been reported; however, relationship to the drug has not been established with certainty: sweet taste, oral and gastric burning, paradoxical swelling, headache, jaundice, thrombophlebitis and emboli and acute pancreatitis.

Lasix (furosemide)-induced diuresis may be accompanied by weakness, fatigue, lightheadedness or dizziness, muscle cramps, thirst, increased perspiration, urinary bladder spasm, and symptoms of urinary frequency.

Dosage and Administration

ADULTS

The usual adult dose of Lasix (furosemide) is 20 to 80 mg given as a single dose.

If the diuretic response with a single dose of 20 to 80 mg is not satisfactory, the following schedule should be used: Increase this dose by increments of 20 or 40 mg not sooner than 6 to 8 hours after the previous dose until the desired diuretic effect has been obtained. This individually determined single dose should then be given once or twice daily. The dose of Lasix (furosemide) may be carefully titrated up to 600 mg per day in those patients with severe clinical edematous states.

With doses exceeding 80 mg/day and given for prolonged periods, careful clinical and laboratory observations are particularly advisable.

Hypertension—The usual dose of Lasix (furosemide) is 40 mg twice daily both for initiation of therapy and for maintenance. Careful observations for changes in blood pressure must be made when this compound is used with other antihypertensive drugs, especially during initial therapy. The dosage of other agents must be reduced by at least 50 percent as soon as Lasix (furosemide) is added to the regimen to prevent excessive drop in blood pressure. As the blood pressure falls under the potentiating effect of Lasix (furosemide), a further reduction in dosage, or even discontinuation, of other antihypertensive drugs may be necessary. It is further recommended, if 40 mg twice daily does not lead to a clinically satisfactory response, to add other hypotensive agents, e.g., reserpine, rather than to increase the dose of Lasix (furosemide).

INFANTS AND CHILDREN

Pediatric Administration: The usual initial dose of oral Lasix in infants and children is 2 mg/kg body weight, given as a single dose. If the diuretic response is not satisfactory after the initial dose, dosage may be increased by 1 or 2 mg/kg not sooner than 6 to 8 hours after the previous dose. Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

How Supplied—Lasix Tablets 40 mg (furosemide) supplied as white, round, monogrammed, scored tablets.

Lasix Tablets 20 mg (furosemide) supplied as white, oval, monogrammed tablets.

Note: Dispense in dark containers. Exposure to light may cause slight discoloration which, however, does not alter potency.

Additional Information

Toxicology

The acute toxicity of Lasix (furosemide) has been determined in mice, rats, and dogs. In all three animal species, the oral LD_{50} of Lasix (furosemide) exceeded 1000 mg/kg of body weight, while the intravenous LD_{50} ranged from 300 to 680 mg/kg. Intragastric injection of the drug in newborn rats resulted in an LD_{50} of 380 mg/kg.

The acute toxicity of high doses of Lasix (furosemide) was characterized by convulsions, paralysis, and collapse. Surviving animals often became dehydrated and depleted of electrolytes due to the diuresis induced by Lasix (furosemide). In the newborn rats, intragastric injection of the drug caused hyperactivity and anorexia.

Chronic toxicity studies with Lasix (furosemide) were done in rats and dogs. In a one-year study in rats, renal tubular degeneration occurred, with all doses higher than 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day). A six-month study in dogs revealed calcification and scarring of the renal parenchyma at all doses above 10 mg/kg (83 percent of the maximal recommended human dose of 600 mg per day).

Reproductive Studies

The effects of Lasix (furosemide) on embryonic and fetal development and on pregnant dams were studied in mice, rats, and rabbits.

Lasix (furosemide) caused unexplained maternal deaths and abortions in the rabbit when 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day) was administered between days 12 to 17 of gestation. In a previous study the lowest dose of only 25 mg/kg (2 times the maximal recommended human dose of 600 mg per day) caused maternal deaths and abortions. In a third study, none of the pregnant rabbits survived a dose of 100 mg/kg. Data from the above studies indicate fetal lethality which can precede maternal deaths.

The results of the mouse study and one of the three rabbit studies also showed an increased incidence of hydronephrosis (distention of the renal pelvis and, in some cases, of the ureters) in fetuses derived from treated dams as compared to the incidence in fetuses from the control group.

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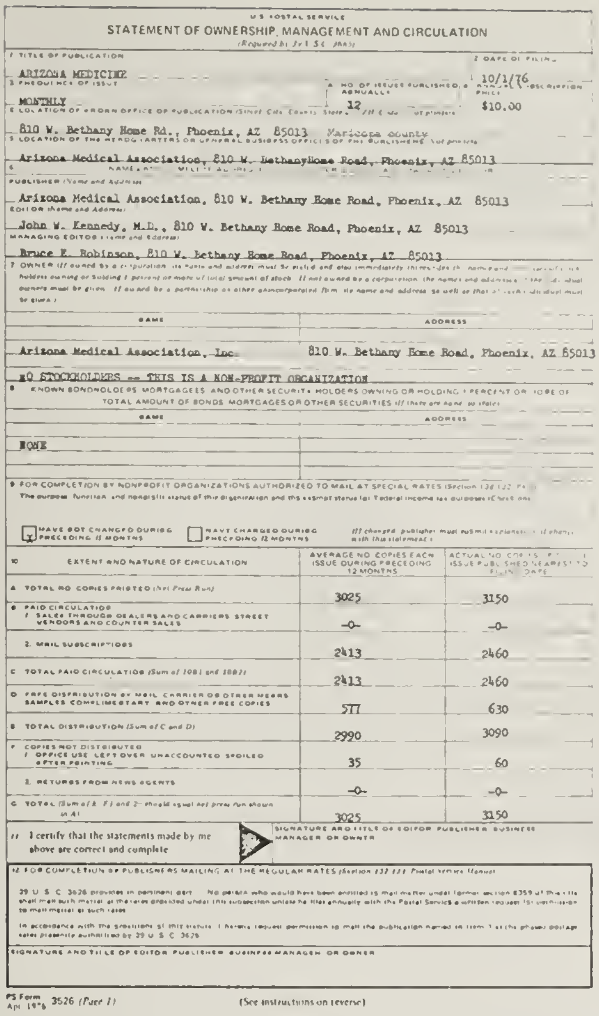

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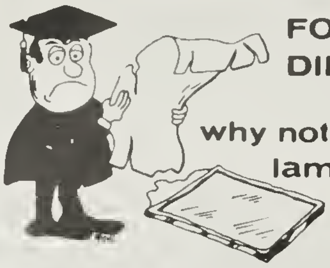

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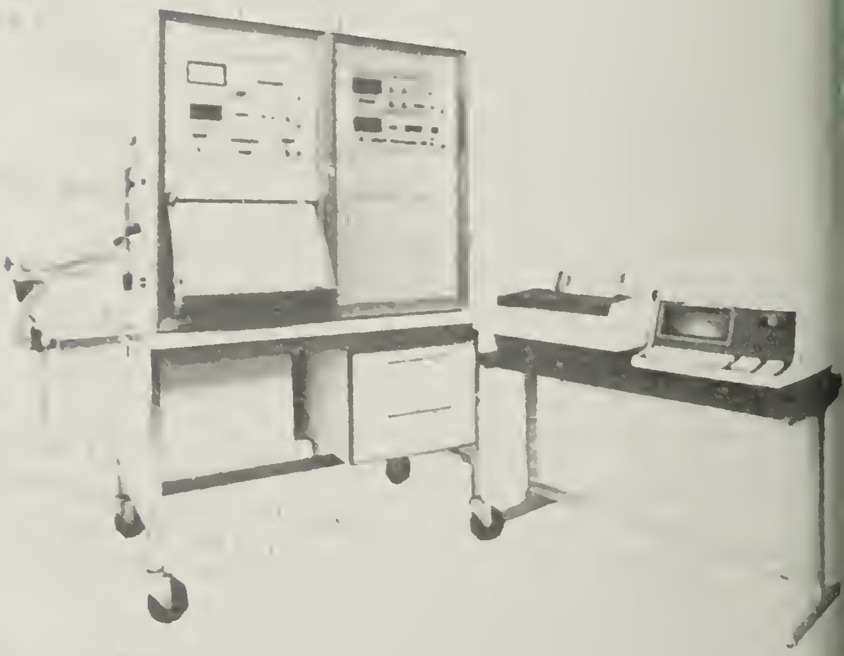

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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful

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Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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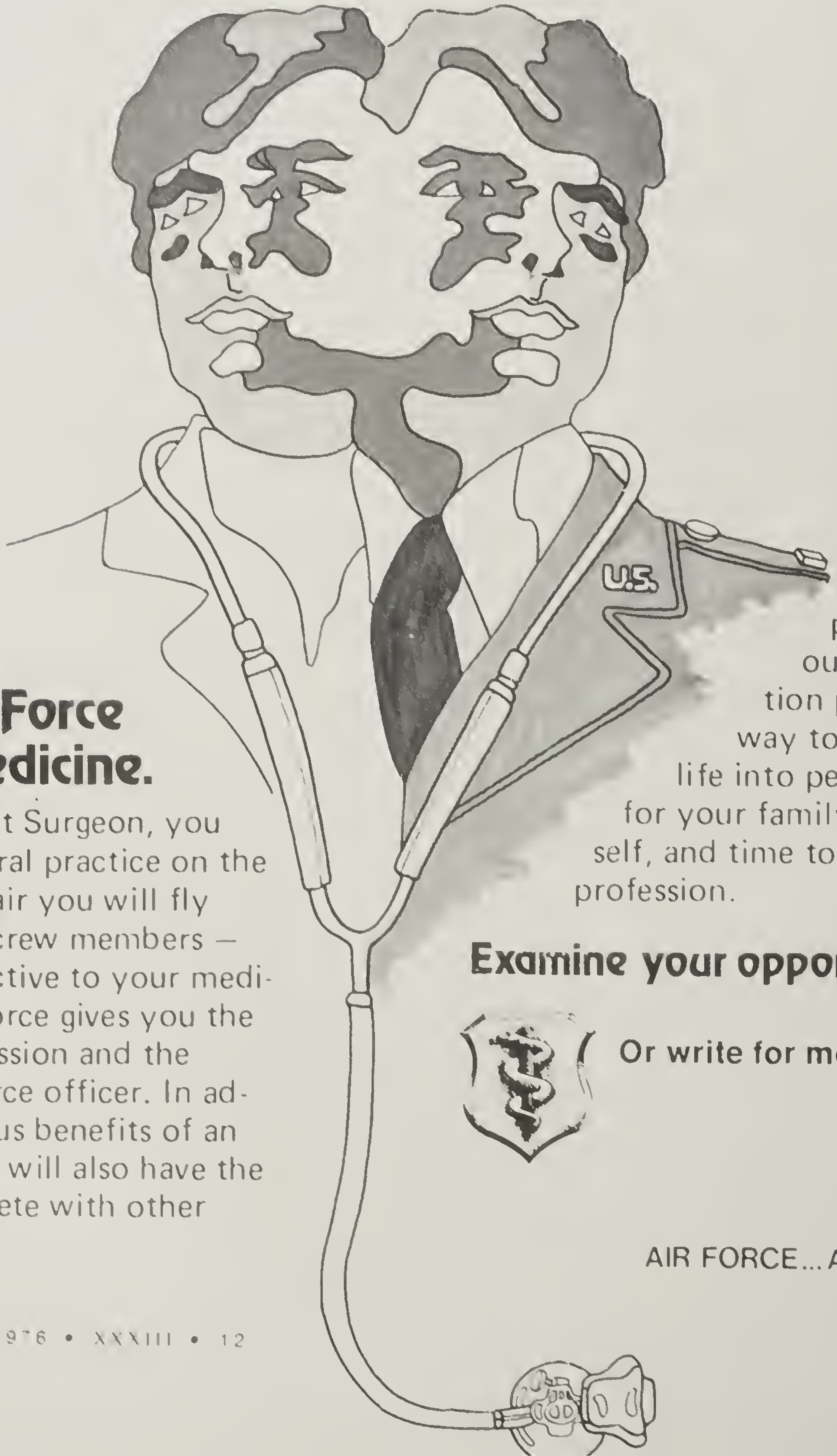

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2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.
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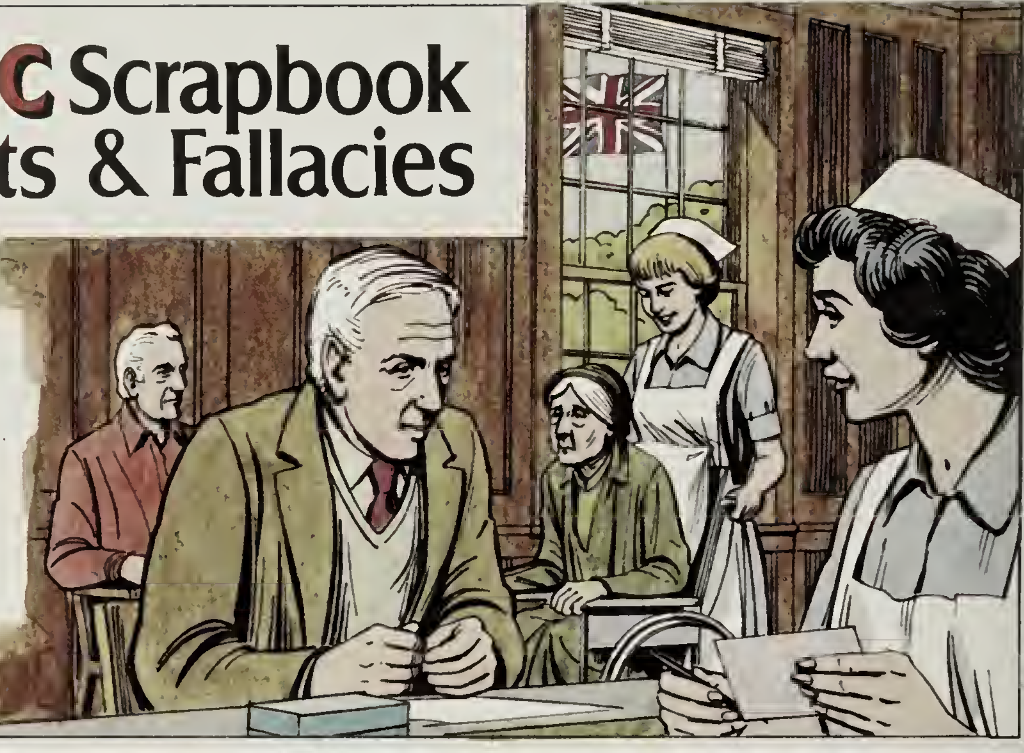

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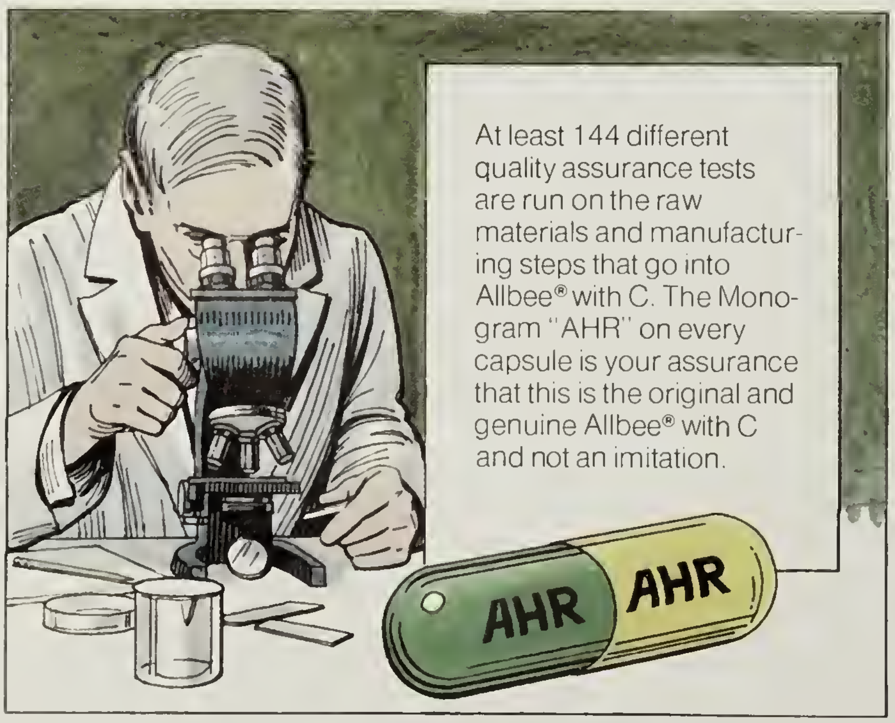

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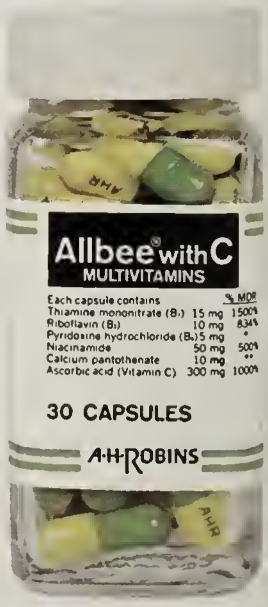
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INFLUENZA AND INFLUENZA IMMUNIZATION

VINCENT A. FULGINITI, M.D.

"This is a timely review of influenza. All physicians should be knowledgeable about a topic having such widespread international implications."

INTRODUCTION

The current interest in influenza virus infections and protection against them by immunization prompted this review. The purpose of this report is to summarize current knowledge of the influenza virus, the disease it produces, the epidemic and pandemic potential and risk and the efficacy and safety of immunization.

The Virus

Influenza virus belongs to the group of myxoviruses. Individual virus particles have a characteristic structure, the components of which have both clinical and immunologic significance.[1]

The core of the virus particle contains a ribonucleoprotein (RNP) which is the genetic unit. Human influenza viruses can be divided into 3 distinct types based upon antigens of the particular RNP; these are termed types A, B and C. Type A viruses are the major human pathogens accounting for sporadic disease, epidemics and pandemics. Asian, Hong Kong, Victoria and Swine "flu" are all caused by A-type influenza viruses. Type B viruses also produce sporadic disease and smaller-scale epidemics. Type C viruses are encountered infrequently in significant human disease.

The RNP core is surrounded by a membrane which contains protein on its inner surface and lipid on its outer surface. The outer lipid is derived from the host cell in which the virus is growing. This is one reason that egg-sensitive humans may react to influenza virus vaccines grown in embryonated eggs.

Attached to the outer membrane are two distinct structures; a hemagglutinin (H) and a neuraminidase (N). These function in attachment of the virus to host cells in the initiation of infection and probably in penetration of the virus into the cell. Only a limited number of different H and N structures have been discovered; approximately 11 H and 7 N proteins. Each type of A virus contains a specific H and a specific N protein, which are used to identify that virus and related strains. Subtle changes in H and N over time result in "antigenic drift", whereby the virus slowly changes its antigenic structure.[2] In general, host response to a specific H or N type provides immunity to similar strains despite these small and subtle changes.

Occasionally, and for reasons which are totally unknown, a major structural change occurs in the H or N protein. This is termed "antigenic shift".[2] The clinical significance of this change lies in the resistance of the "new" virus to immunity developed against previous strains. The "shift" to new structural H or N types provides an entire population which has not been exposed to it, or related strains. Most major pandemics and the tailing epidemics have occured following such a major influenzal structural alteration.

Some of the characteristic virus strains are indicated in Table 1 and characterized by their distinctive H and N types.

Immune Response to Influenza Virus

Influenza virus evokes several immune responses in man. The best studied is the circulating antibody to various viral components.[3] Antibody has been measured against RNP, H and N among other components. Three serologic tests have been employed most commonly; complement fixation (RNP antibody), hemagglutination-inhibition and neutralization (antibody against the H antigens). There are newer techniques measuring antibody against the N antigens and other protein antigens in the core and inner membrane of the virus. The anti-neuraminidase antibody may be an important protective factor in immunity.

It has long been believed that immunity to influenza was directly related to the specific type and quantity of antibody evoked by natural infection or immunization. Some investigators believe that a level of antibody of 40 or higher (the reciprocal of the dilution of serum inhibiting the effects of some component of the virus, hence 1/40 = 40) provides protection against wild

Submitted for publication 10/76.

From: Dept. of Pediatrics, Arizona Medical Center, Tucson AZ 85724.

Table 1

Some major types of A influenza virus with their clinical significance and hemagglutinin (H) and neuraminidose (N) structure

Virus Designation*	Clinical Comment	Type H	Type N
A/PR/8/34	Probable descendant of "swine" virus prevalent in 1920's & 30's	0	1
A/FM/1/47	Not true "shift"—no pandemic	1	1
A/JAPAN/305/57	The "Asian flu"—cause of 57-58 pandemic	2	2
A/HONG KONG/1/68	The "Hong Kong" flu—cause of 68 pandemic	3	2
A/VICTORIA/3/75	Descendent of A Hong Kong— current 75-76 epidemics	3	2
A/NEW JERSEY/8/76	Fort Dix isolate in 1976	sw1	1

*By convention, influenza viruses are names as follows: Major type/geographic origin/strain #/year isolated (HN) types

PR = Puerto Rico FM = Fort Monmouth

virus of the same or similar type.[3] Recent immunologic investigations have clouded the picture. Secretory antibody of the IgA sub-type may play a significant role in protection, irrespective of the presence and height of the serum antibody level.[4] There is even some speculation that non-antibody mechanisms such as cell-mediated immunity linked to circulatory or local small lymphocytes may be important.[5]

At best the immunology of influenza virus infection is imperfectly understood. For reasons of immunization objectives and pademic potential, I shall take the conventional view that immunity is linked to the presence of serum antibody, the only practical viewpoint at present.

The Disease

Unfortunately, the label "influenza" has been applied too broadly to a large spectrum of diseases, not all of which are due to infection with influenza virus. The following discussion is limited to illness definitely associated with influenza virus. Physicians must be cautious in their nomenclature since efficacy of vaccine and public perception may depend upon precise identification of the true influenzal illness. Folklore, lay and medical, has included many respiratory, gastrointestinal and more diffuse illnesses in the terms "flu" or "grippe", falsely expanding the role of influenza virus in disease production and clouding interpretation of vaccine efficacy.

The classic influenzal illness has a characteristic respiratory and systemic symptomatology.[6,7]

Following a short (1-3 days, usually) incubation period, abrupt onset of fever, chills, episodic sweating, lassitude, malaise, headache and myalgia. Few infectious agents produce a "snicker" or more "toxic" patient than does influenza virus. Occasionally, there are prodromal respiratory symptoms of minor degree.

The illness may be variable in severity and in the mix of symptoms. Some patients manifest little more than the above constitutional pattern. Others will develop a dry cough, associated with severe substernal chest pains often of considerable severity. Conjunctivitis and nasal stuffiness with little drainage also occur frequently.

Contrary to popular belief, gastrointestinal symptoms are few. Some patients will experience vomiting and most are anorexic but diarrhea is rare, as is abdominal pain.

The illness may be frustrating with profound lethargy and severe symptoms. It can be much milder. Symptoms often persist, especially weakness, myalgia and painful cough. Some individuals complain of prolonged weakness, often lasting weeks, the so-called influenzal "asthenia".

Children undergo much less severe illness, as a rule. However, severe pneumonia, bronchiolitis, croup and other respiratory syndromes are encountered during an influenzal epidemic.[8]

Although the majority of patients recover, a significant number have illnesses complicated by bacterial infections. Pandemic influenza is particularly associated with excessive morbidity and death because of the large numbers of persons infected. Even if there is an attack rate of only 20-30%, when one is considering the entire world population, the number of persons ill and the absolute number of deaths is staggering.

The most commonly encountered complications in epidemics or pandemics are respiratory bacterial superinfections and congestive heart failure. These occur with much greater frequency among patients with preexisting respiratory, cardiac and metabolic disease, and also among pregnant women.

Bacterial superinfection has varied in type between pademics.[9] In 1918 the principal agents were *Hemophilus influenzae* and hemolytic streptococci. In the epidemics of the 50's and 60's, *Staphylococcus aureus* has been the predominant agent. Much, but not all, of the morbidity and death were secondary to bacterial infection. Some patients suffered from primary influenzal pneumonia of lethal degree, uncomplicated by bacterial superinfection.

Congestive heart failure is frequent, especially in those with valvular heart disease and in pregnant women. Rarely influenza virus will produce myocarditis and pericarditis. Most often the congestive heart failure is secondary to pulmonary disease or the severity of systemic manifestations.

Although described in epidemics and pandemics, neurologic complications, including encephalitis are uncommon to rare in occurrence. Again the sheer numbers involved in pandemics results in "rare" complications being more prominent.

The Epidemic and the Pandemic Potential

More than any other agent in modern times, influenza A and B viruses have the capacity to infect large populations in a short period of time (epidemics); only influenza A produces worldwide epidemics or pandemics.[2,10] Influenza B produces localized epidemics.

Epidemics appear to result from sufficient antigenic drift (changes in H or N antigenic structure) so that a significant proportion of the population have had little experience with that particular configuration. Other factors also play a role in a poorly-understood fashion. Environmental factors contributing to spread of the virus, to lowered resistance to infection and to respiratory susceptibility have been thought to play a role.

Pandemics occur when influenza A virus undergoes antigenic shift. This structural change results in almost all of the population becoming susceptible by virtue of *no* experience with that antigen or even closely related antigens. The major pandemic strains in recent times (A Asian and A Hong Kong) are examples of such major structural alteration. The interval between pandemics usually results in very few individuals who have had any experience with the strain or a related virus. For example, should A New Jersey (the current Swine-like virus) develop into a pandemic virus, only individuals older than 55 years would have had substantial experience with a related virus. In this age group many such individuals have serum antibody which neutralizes A New Jersey. This antibody stems from infection with similar influenza viruses some 40 years ago. Individuals younger than 55 do not possess such antibody since related strains have not been present in the interval.

The current epidemiologic view is that the same H and N types tend to recur at long intervals in human history.[12] For example, it is widely held (based upon serologic studies) that the late 19th century strains were similar to the A Asian virus of 1957-58 and that A Hong Kong H-related viruses were prevalent in the early 20th century. Extension of this belief gives credence to the idea that swine-like strains will probably reappear soon after an almost 60 year hiatus.

The concern evoked by discovery of an antigenic shift in H type is well-founded, since in modern virologic times serious pandemics have followed.

Swine-influenza—1976

Swine-like influenza viruses (Hsw type) have been encountered rarely since modern virology permitted identification of antigenic types.[13] In almost all of these rare identifications, the infected human has had contact with hogs, directly or indirectly. Prior to 1976 and since 1934, when influenza virus was first isolated, no human to human transmission of Swine-type influenza virus has been observed.

With these facts in mind, one can better understand the wave of concern that rolled through the scientific community when an investigation of the death of a recruit at Fort Dix in February 1976 implicated a Swine-like virus.[14] After verification of the identity of the agent, obtained from the lungs of the dead recruit, further investigation resulted in identification of 4 other swine-like isolates from sick recruits and antibody in a large number of recruits, their families and other contacts. In contrast, serologic review of populations and cases of influenza from across the U.S. and in other parts of the world failed to identify significant numbers of individuals with antibody. A few were identified but in all instances these were isolated findings and unassociated with spread.

Extended investigation uncovered a few instances of actual swine-like influenza virus infection. Each of these were isolated and related to contact with hogs. One fatality was recorded in a young man with Hodgkins disease at the Mayo Clinic.[15]

Limited population surveys organized by the Center for Disease Control have indicated that only individuals older than 55 years have significant antibody to swine virus, suggesting that they experienced infection in childhood with related strains.[11] Much later, as an unexpected result of vaccine trials, experience with swine-related viruses in the 24 years and older group is suspected.[16] These individuals responded to influenza vaccine with an excellent antibody titer and with minimal reaction, suggestive of prior experience.

As a result of the preliminary data collected by mid March, knowledgeable virologists, physicians and public health officials recommended that an attempt be made to manufacture, test and administer influenza vaccine to as many as 210,000,000 citizens.[17] The major impetus of the time was the strong possibility that the Fort Dix episode was a harbinger of a major pandemic. This recommendation was accepted by the President and Congress initiated implementation by appropriating sufficient funds to develop, manufacture and test swine-flu vaccine prepared from the A/New Jersey/8/76 (H_{sw1} N_1) strain.

Between March and September the following significant events have occurred:

(1) Four manufacturers have produced lots of two different types of swine vaccine (to be discussed later).

(2) Elaborate and careful field testing began and is still underway. The objectives are to determine antigenicity and safety (reactogenicity), of the individual vaccines. The optimal dosage, schedule and age-appropriate recommendations are also being determined.

(3) Scientific advisory groups were identified and assembled periodically to review both the field experience with natural swine influenza infections and the results of immunization trials, as they become available.

(4) The issue of liability for intrinsic risk, if any, from influenza vaccine administration was addressed. The drug manufacturers did not wish to release any vaccine unless they were either insured against such risks or "held harmless". The insurance companies failed to identify a suitable coverage plan, despite urgings from top governmental officials. Finally, in order to insure progression of the immunization program, Congress enacted Public Law 94380 on August 10, 1976 which provided for the U.S. to be the guarantor of intrinsic risk claims.[18] Several specific provisions are of interest to physicians. In order to be protected from such suits a participating physician must: (1) charge no fee for the vaccine or its administration, (2) use the federal form for obtaining consent of the patient or parent to be immunized.

(5) Public Health officials at the Center for Disease Control (CDC) have conducted meetings with each state's health authorities to establish the method for distribution and administration of the vaccine when available.

Influenza Vaccine

The first attempt at immunization against influenza occurred in the 1940's.[19] The vaccines used were prepared in the allantoic cavity of embryonated hen's eggs. The finished product probably contained a great deal of extraneous material derived from the eggs in which the virus was grown.

Early studies of efficacy demonstrated inconsistent results from trial to trial. It is now believed these inconsistencies were related to variable incidence of influenza, to variable potency of the vaccines utilized and to variation in antigenicity of prevalent and vaccine strains.

These products evoked marked local and systemic reactions believed due to both intrinsic influenza virus toxicity and to extraneous materials carried along from the eggs in which the virus was grown. For many years killed influenza vaccine has had the reputation of being so reactogenic as to prohibit its use in routine practice. Instead, it has been recommended for use only in individuals with chronic diseases who are at high risk and in epidemics or pandemics.

Technological advances in influenza virus vaccine manufacture have greatly reduced reactogenicity when compared to early vaccines.[20] Improved clarification, separation and centrifugation methods have resulted in more purified products with far less extraneous material.

A second major advance was the discovery that the H (and N) antigens evoked antibody which could protect against infection.[21] Several methods have been devised to disrupt the virus particle. Tween-80 and ether treatment, deoxycholate and tri-(n butyl)-phosphate disruption produce vaccines which are rich in H and N antigens and their associated membranes but which can be freed from the core antigens and other proteins believed the cause of some vaccine reactions; (these are called "split-product" vaccines).[22]

Since their development influenza virus vaccines have been controversial; partially because of the reactogenicity referred to above, and partially because of variable efficacy in clinical trials. Without reviewing the elaborate history of this controversy, it is pertinent to summarize current consensus.

(1) Inactivated influenza virus vaccines are of variable potency. The commonly-used measure of potency, chick-cell-agglutination units (CCA), is now believed not to represent a precise quantitation of antigenic content. No other marker of potency has emerged.

(2) Given a whole virus inactivated vaccine of sufficient potency, antibody, and protection against disease, will be provided to 70% or more of recipients, on the average. Some experts estimate less protection, and some more.

(3) Immunity appears relatively short-lived with booster doses necessary every year.

(4) The degree to which the antigenic content of vaccine matches the H and N configuration of current influenza strains is a major determinant of the degree of protection.

(5) The split-product vaccines (disrupted virus high in H and N antigens) are generally less effective in evoking serum antibody but have much less reactogenicity.

(6) The dosage of vaccine depends on the potency of a particular lot. Recommendations usually include a specific minimal CCA unitage to be used per dose.

(7) The only effective route for administration of inactivated influenza vaccines currently in use is subcutaneous and intramuscular. The intradermal route should not be employed.

Side-effects and Adverse Reactions

The usual side-effects in adults to inactivated whole virus influenza vaccines are self-limited. They consist of fever, headache, myalgia, malaise and local signs of irritation or reaction at the injection site (tenderness or soreness, induration and erythema). These usually occur from 6-24 hours post-immunization and rarely last longer than 24 hours.

More severe adverse reactions are rarely encountered and are poorly documented as being specifically caused by influenza immunization. Among reactions reported in temporal association with vaccine administration are: a variety of neurological syndromes including encephalopathy, myelopathy, radiculopathy and polyneuropathy; collapse; hyperpyretic syndrome. There are only a handful of such reports.

The current field trials with whole-virus vaccine in adults indicate a very low reaction rate in the group older than 24 years—overall less than 2%.[16] There was some variability among the specific vaccines tested. In individuals between 18-24 years the reaction rate was slightly higher—less than 5%. In both groups split-product vaccines produced many fewer reactions.

Children have usually experienced a higher reaction rate than adults. In addition, in two recently published studies, whole virus vaccines of both A and B types (not the swine-flu vaccines) produced febrile convulsions in a few infants and children under the age of 3 years.

Vaccine trials with the A/New Jersey/76 virus document the increased reaction rate in children.[16] As many as 15-20% reacted to whole-virus vaccine, some with moderately severe reactions, but none with convulsions or lasting effects. In contrast, split-product vaccine produced many fewer reactions of little or no severity, and little different from placebo recipients.

Inactivated influenza vaccine is produced in hen's eggs. There are two ways in which egg antigens, probably lipids and proteins, are incorporated into the final vaccine. First, the virus in reproducing in egg cells utilize the egg-cell membrans as part of its outer coat. In addition, non-viral egg antigens are carried forward during the harvesting of the virus and are not completely eliminated in the purification process. Rarely, an individual who is hypersensitive to egg antigens may experience an untoward reaction to the vaccine. Two such instances occurred during the recent swine-influenza vaccine studies.[16] There is no reliable way to predict which egg-sensitive persons will react. Therefore, the vaccine should not be administered to such individuals.

Most influenza virus vaccines have a preservative added in the manufacturing process. This usually is a mercuric compound. Rarely, a person with known mercury sensitivity may react to the vaccine.

Recommendations for 1976-77[24, 25, 26]

Although all of the data are not yet available the following recommendations have been promulgated.

(1) Whole virus; inactivated influenza A/New Jersey/76 (Hsw$_1$ N$_1$) vaccine should be administered as a single intramuscular dose of 200 CCA units to all individuals 25 years of age or older.

(2) Individuals between 18 and 24 years of age should receive one dose of the same vaccine. A second dose may be required separated by at least 4 weeks from the first dose. Current field trials will provide an answer.

(3) High-risk adults who have chronic diseases should receive bivalent A Victoria 3 75 (H_3 N_2) and A New Jersey 8 76 (Hsw_1 N_1) whole virus vaccine containing 200 200 CCA units. One dose should be administered intramuscularly.

(4) High-risk children 3 years of age and older (to age 18) with chronic diseases should receive the bivalent A Victoria 3 75 (H_3N_2) and A New Jersey 8 76 (Hsw_1 N_1) inactivated vaccine of the *split-product type* containing 200 200 CCA units. A A two-dose schedule is mandatory with a minimum interval of 4 weeks between the doses.

(5) Insufficient data are available to make any recommendation concerning immunization of healthy children between the ages of 3 years and 18 years. On Nov. 23 the American Academy of Pediatrics Committee on Infectious Diseases extended its earlier recommendation as follows:
Normal Children 3-18 years — Two doses of split product monovalent A NJ 8 76, 0.5ml (200 CCA units) 4 weeks apart.

(6) At the present time no vaccine should be administered to children less than 3 years of age, regardless of their health or disease state. 6 months - 3 years — Insufficient experience to make a recommendation.

(7) Pregnant women should be considered as having the same benefits and risks as the age group to which they belong.

In all instances of recommended influenza immunization the following features should be noted:

(1) Any physician or delegate who participates in influenza immunizations whether in his her office or other sites should charge *no fee* for the vaccine or its administration if liability coverage by the federal government under Public Law 94380 is desired. The vaccine will be provided at no cost. Physicians who charge a fee for vaccine administration cannot qualify for federal liability protection; they should consult their own carrier to be certain that the usual coverage applies.

(2) All persons immunized should be fully informed as to the benefits and hazards of vaccine administration and to the risks of influenza virus infection. To qualify for liability-coverage by the federal government, physicians must use the standard federal form which is appropriate to the age group of the recipient and to the vaccine to be administered. These forms will be distributed with the vaccine by State and local Health Departments. Prior to receipt of the vaccine the patient or guardian is required to sign the form indicating that:
   (a) the form has been read
   (b) opportunities to answer all questions have been afforded
   (c) he she requests the vaccine for himself herself or for a person for whom they are the legal guardian.

(3) No individual who is egg sensitive should receive this vaccine as it is prepared in hen's eggs and may contain egg protein which could cause a reaction in such recipients. Some feel that an intradermal test with the vaccine should be employed in this group; if positive no vaccine should be given, if negative cautious administration may be attempted.

(4) All high risk individuals 3 years of age or older should also receive Influenza B vaccine as they are also susceptible to more severe disease from this agent than are healthy individuals. The manufacturer's package insert should be consulted for specific dosage recommendations.

Treatment of Influenza

General: Patients with moderate or severe influenza will confine themselves to bed. All patients with symptoms producing fatigue should be cautioned as to the need for rest and slow resumption of normal activities.

During the acute phase appropriate measures for reduction of fever (antipyretics and fluids) and reduction of pain (analgesia and cough suppressants) should be employed.

Specific:

Amantadine hydrochloride has been proved to be an effective prophylactic against Influenza type A infections.[27,28] To a limited extent it may have some therapeutic usefulness. There are serious pharmacologic problems with use of amantadine that has resulted in some experts suggesting it not be used widely. Dr. George

Jackson, an experienced virologist and clinician, has endorsed its use clinically and feels some of the concerns may be overstated.[27]

Amantadine is a primary amine which has been synthesized in its HCL form. Its pertinent action in influenza appears to be blockade of cells to penetration by influenza virus. The exact mechanism is not known.

At usual doses in otherwise healthy recipients it appears non-toxic for most people. However, the therapeutic toxic margin is close and doses in excess of those recommended can result in distrubances in sensorium, changes in heart rate, slight diuresis and cough. Recipients of high dosages may be excited, anxious and demonstrate other signs of CNS stimulation. Even a few persons at recommended doses may develop symptoms. In some patients drowsiness and sleepiness predominate, i.e. a CNS depressant effect.

Elderly persons and possibly those with pre-existing CNS disease may experience greater susceptibility to the CNS side-effects of this agent.

The usual adult dose is 200-300 mgm day. When used prophylactically it must be administered prior to exposure and continued during the entire period of exposure. This necessity makes use of Amatadine impractical for the general population. Coupled with some scientists' scepticism as to the significance of clinical efficacy data, Amatadine has been little used and not often recommended.

Data are scant for use of this agent in children.[29,30] The following recommendations are those of the manufacturer. Many experts suggest that this drug not be used in children.

Under 1 year of age—Insufficient data. Do not administer.

1-9 years of age—Do not exceed 150 mgm day. Give 2-4 mgm pound of body weight day in 2-3 divided doses.

9-12 years of age—Do not exceed 200 mgm. Give 100 mgm twice daily.

Amantadine should not be administered to pregnant women or to patients at any age who have significant CNS deterioration. The latter caution is especially pertinent among the elderly and for prolonged dosage. Some experts do not accept the pediatric use of this agent.

If used Amatadine should be given for at least 10 days after a single exposure and for periods up to 90 days for continuous or repetitive exposure.

No other agent can influence the course of

Table 2

SUMMARY OF CURRENT RECOMMENDATIONS FOR INFLUENZA IMMUNIZATION

Age Group	Health Classification	SUBCUTANEOUS INACTIVATED VACCINE*		
		Monovalent Whole-Virus	**Bivalent Whole-Virus**	**Bivalent Split-Product**
5 yrs. & older	Normal	One-dose	—	—
5 yrs. & older	Chronic Disease**	—	One dose	—
	Pregnancy***	One-dose	—	—
18-24 years†	Normal	One dose	—	—
	Chronic Disease**	—	One dose	—
	Pregnancy***	One dose	—	—
3 yrs. to 18 yrs.	Healthy		NO RECOMMENDATION YET	
	Chronic Disease**	—	—	Two doses
Less than 3 yrs.	Healthy		NOT TO RECEIVE VACCINE AT PRESENT TIME	
	Chronic Disease			

*Monovalent Whole Virus = A New Jersey 8 76 (Hsw$_1$ N$_2$) 200 CCA units
Bivalent Whole Virus = A New Jersey 8 76 (Hsw$_1$N$_1$) plus
A Victoria 3 75(H$_3$ N$_2$) 200 200 CCA units
Bivalent Split-Product — A New Jersey 8 76(Hsw$_1$ N$_1$) plus
A Victoria 3 75 (H$_3$N$_2$) 200 200 CCA units

**Chronic disease includes: chronic bronchopulmonary disorders; chronic cardiovascular disorders, especially valvular heart disease; chronic metabolic disease, especially diabetes; chronic renal disease, chronic neuromuscular disease and special groups including those with malignancies, particularly of the lymphatic system, including leukemia and Hodgkins disease; those receiving immunosuppressive therapy; and those with congenital immunodeficiencies.

***Pregnancy is not a contraindication to influenza vaccine.

† Recommendations for this group may change. *High Risk Children or Chronic Disease* 3 - 18 years — as stated in table. 6 months - 36 months — two doses of split product bivalent A NJ 8 76 and A Victoria 3-75). 0 25 ml (100 CCA units of each component) at least 4 weeks apart.

present influenza despite anecdotal reports and folklore, lay and medical.

For bacterial complications, an appropriate antibiotic regimen should be employed. It is difficult to predict which bacteria will predominate if we experience a major influenzal pandemic. In 1918 the streptococci and *Hemphilus influenzae* were most prominent. In the 1957 and 1968 outbreaks *Staphylococcus aureus* caused most of the complicating infections. In 1977 the Staphylococcus has become less prominent but one suspects that it and Hemophilus are likely to be encountered in large numbers. The physician should be guided by common experiences in the event of an outbreak.

SUMMARY

Influenza virus characteristics, immunity to influenza, manifestation of the disease, and prevention and treatment are reviewed in this report. In addition a brief summary of the history of the occurrence of swine-like influenza virus infections in 1976 is presented. Current recommendations for influenza immunization are presented along with the necessary steps a physician must take to qualify for liability coverage for intrinsic risk by the federal government.

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CONGENITAL MESOBLASTIC NEPHROMA OF INFANCY

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ALEX NEWMAN, M.D.
FELIPE U. LIM, M.D.

> **"An accurate diagnosis and understanding of the mesoblastic nephroma, previously known as congenital Wilm's tumor, allows the physician to withhold radiation and chemotherapy and the complications so commonly encountered from these modes of therapy."**

Most neonatal renal tumors are the congenital mesoblastic nephromas of infancy; these are benign and represent mesenchymal hamartomatous maldevelopment of the fetal kidney. This group of tumors has only relatively recently been separated from the malignant Wilms' tumors. The purpose of this paper is to report two recently diagnosed cases of this entity and to emphasize its differentiation from Wilms' tumor.

REPORT OF CASES

Case 1. B.B.D. A newborn full term male was found to have a right flank mass on palpation. The mass was hard, not fluctuant, and not connected to the liver. Urographic studies showed a large mass in the lower pole of the right kidney displacing upwards and distorting the renal pelvis and calyces, and displacing down-

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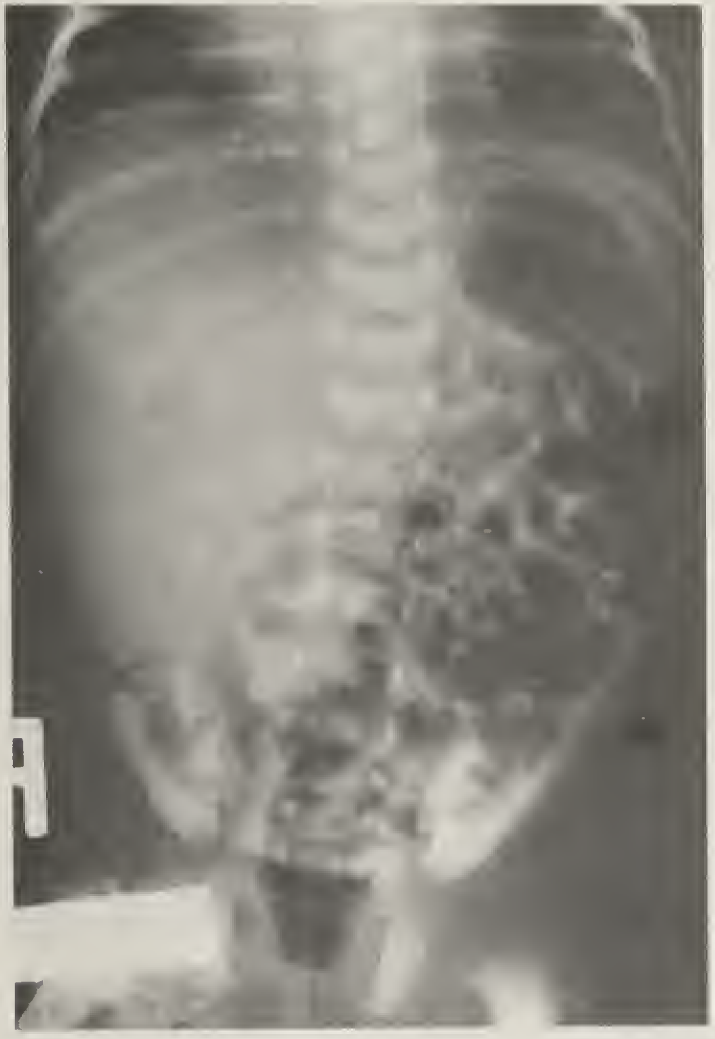

Figure 1

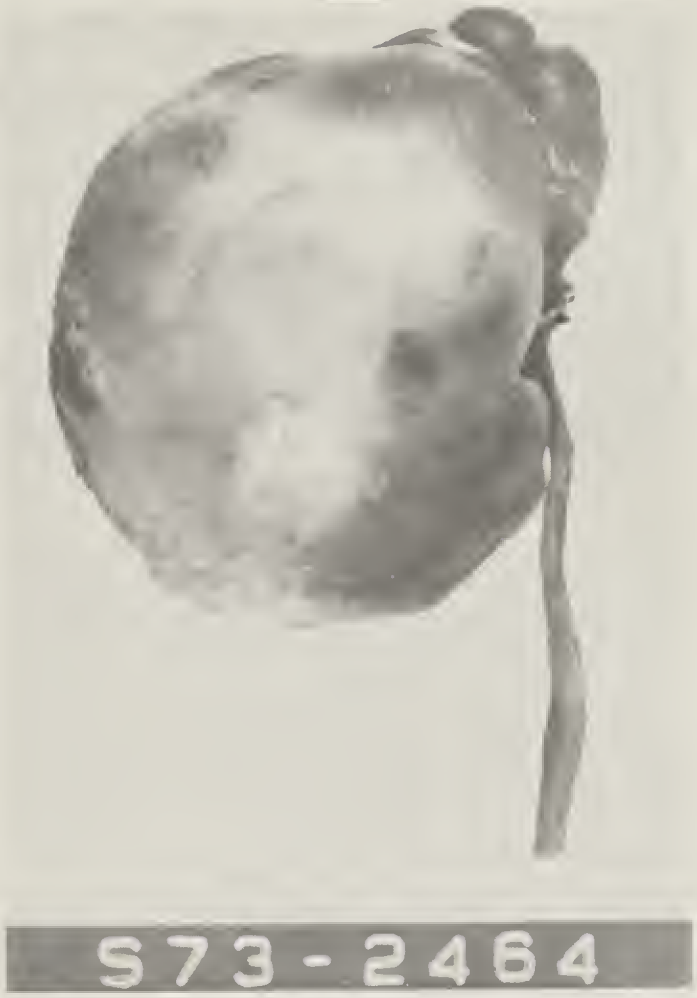

Figure 2

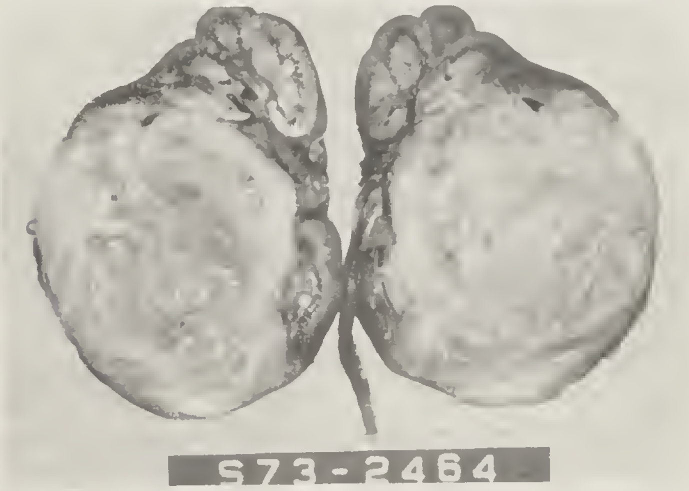

Figure 3

wards and anteriorly the adjacent loops of bowel (Fig. 1). When the infant was 3 days old, laparotomy was performed, and a solid renal tumor was found. Right nephrectomy was performed. The kidney weighed 77 grams and measured 6.5 x 4.8 x 4.6 cms. The tumor mass measured 6.0 x 4.6 x 4.2 cms. and practically replaced the entire lower pole (Fig. 2). It was well circumscribed but not encapsulated. It consisted of whorled strands of pinkish tan rubbery tissue. The tumor resembled a fibromyoma of the uterus (Fig. 3).

Microscopically, the tumor consisted of bundles of spindle cells arranged in a somewhat whorled fashion. The cells exhibited slightly plump nuclei showing no atypism. The ground substance was fibrillar and eosinophilic. Scattered through the lesion, particularly between bundles of tumor cells, were occasional tubular and glomerular elements (Fig. 4), but these appeared to be trapped by tumor rather than present as tumor elements. Trichrome stains revealed the cells to be predominantly fibrocystic or fibroblastic. Normal mitotic figures were noted (Fig. 5). The pathologic diagnosis was congenital mesoblastic nephroma of infancy. Recovery was uneventful, and the patient is alive and well after 3 years.

Case 2. J.A.A. A white female infant was admitted to the hospital at two months of age. A left-sided abdominal mass was felt two weeks prior to admission, and the infant was referred to the hospital as a case of probable Wilms' tumor. Excretory urography (Figs. 6A and 6B) showed a left-sided mass that depressed and distorted the renal elements, and in all ways was roentgeno-

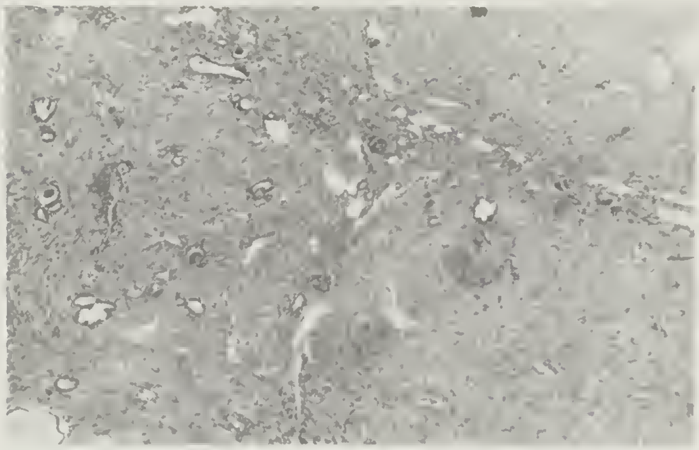

Figure 4

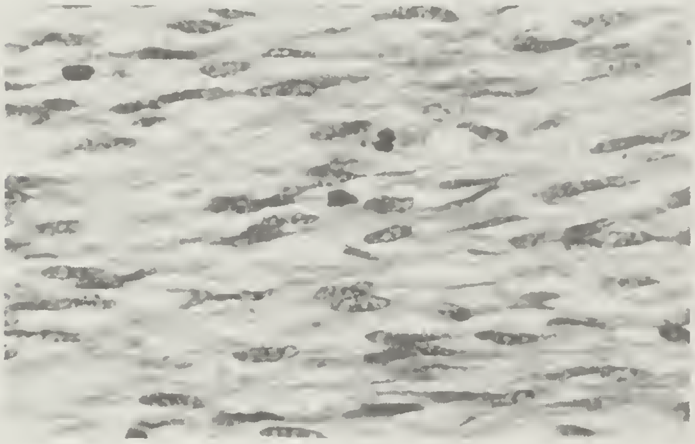

Figure 5

graphically indistinguishable from the findings usually noted in cases of Wilms' tumor. Left nephrectomy was performed and confirmed the presence of left renal neoplasm. The kidney with the tumor weighed 115 grams. The upper pole was replaced by a well circumscribed mass, 5.5 cms. in diameter. It was composed of firm, whorled, glistening, gray-pink tissue throughout and appeared to be well demarcated from the adjacent renal parenchyma.

Microscopically, the appearance of the tumor was essentially similar to the first case. Glomeruli and tubules were surrounded by tumor cells. An added feature was the presence of foci of squamous metaplasia (Fig. 7). No mitotic figures were noted (Fig. 8). The pathologic diagnosis was congenital mesoblastic nephroma of infancy. Recovery was uneventful, and patient is alive and well after one year.

DISCUSSION

Bolande et al.[1] adopted the name "congenital mesoblastic nephroma in infancy" to describe a benign fibromatoid tumor of the kidney which in the past had been confused with Wilms' tumor. This term was actually a modification of

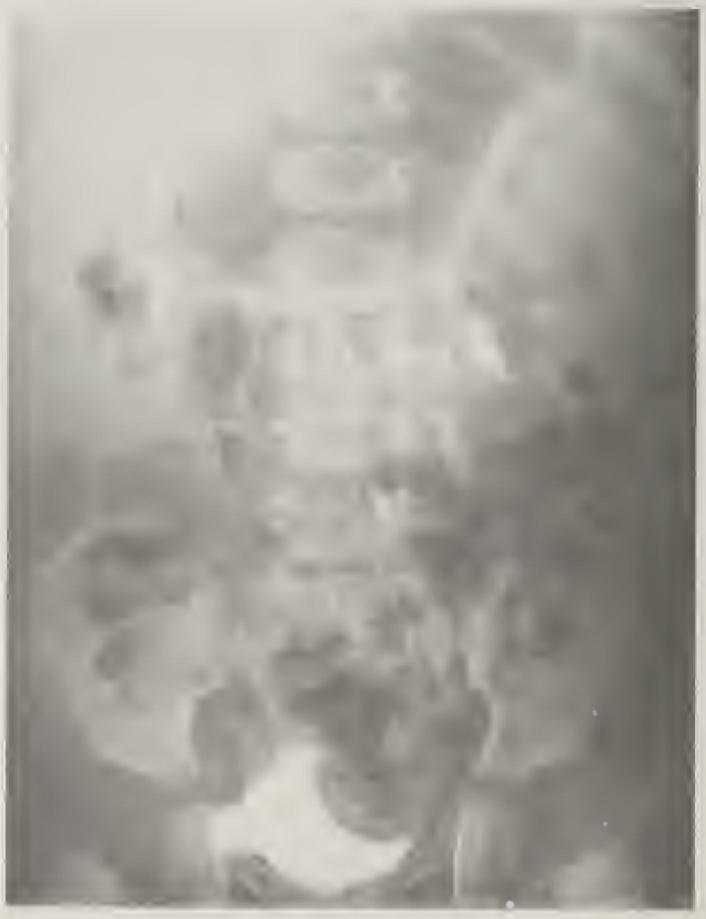

Figure 6a

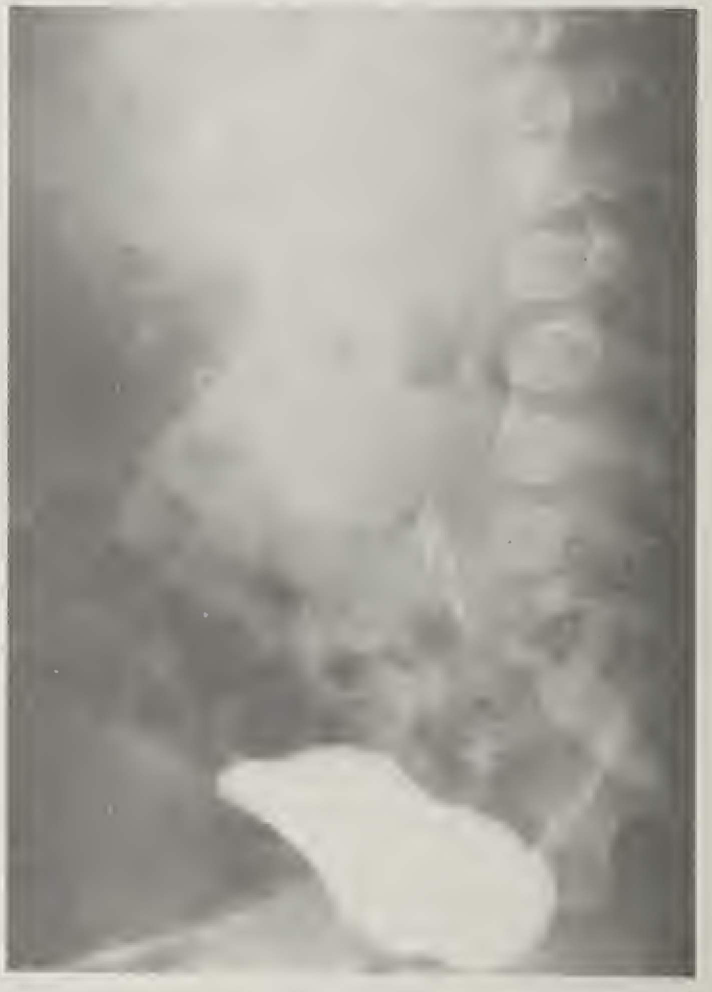

Figure 6b

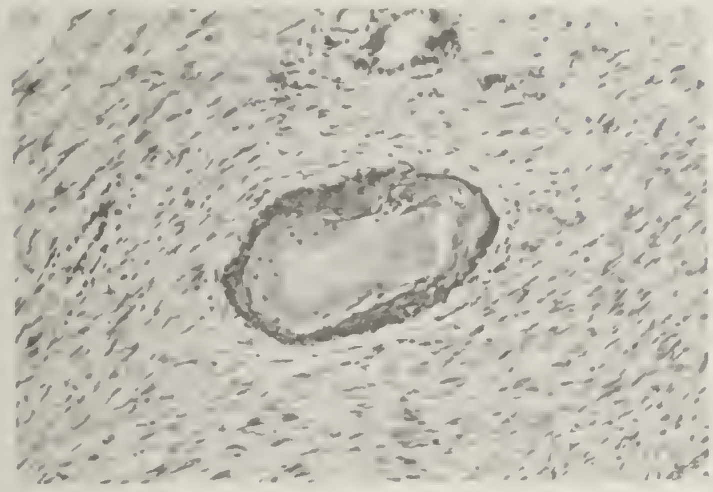

Figure 7

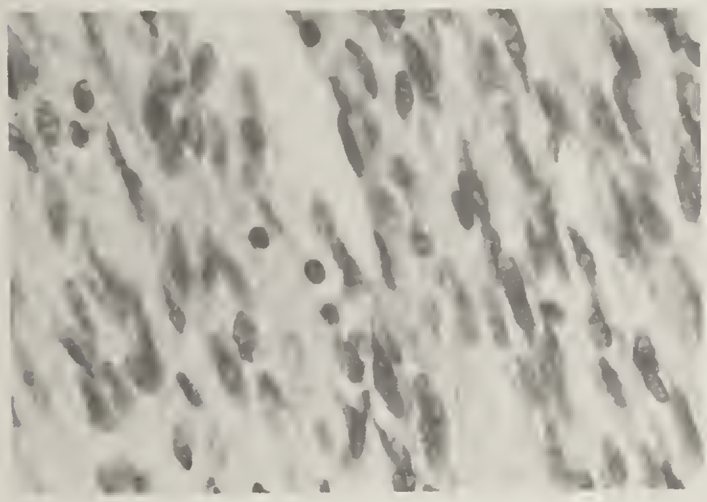

Figure 8

the one coined earlier by Culp and Hartman[2] to describe a class of embryonal, Wilms'-like malignant cortical tumors of the adult kidney, namely the mesoblastic nephroma in adults.

This tumor has been reported under a variety of different labels beginning in 1921 with Kastner[3] who characterized this neoplasm as a spindle-cell sarcoma. Subsequently, this neoplasm has been reported as fibrosarcoma,[4] leiomyosarcoma,[5 6] fibroma,[7 8] leiomyomatus hamartoma,[9 10] which is still the name of choice at the Armed Forces Institute of Pathology, mesenchymal hamartoma,*11* fetal mesenchymal hamartoma,[12] fetal renal hamartoma[13 14] and congenital Wilms' tumor.[15] Indeed, most of the cases reported before 1966 were grouped as congenital Wilms' tumor of the conventional type.

We prefer to continue use of the name, congenital mesoblastic nephroma, because it conveys the idea of its preponderant mesenchymal constituency and of a relationship to Wilms' tumor, while still distinguishing it from that tumor. Above all, the terms, neonatal Wilms' tumor and benign Wilms' tumor used at times to describe this entity are to be emphatically discouraged.

Wilms' tumor is an encapsulated malignant neoplasm with epithelial components. Mesoblastic nephroma is not encapsulated, is firm and rubbery, and rarely shows necrosis or hemorrhage. It is a collagen-rich tumor with no epithelial components; indeed, it is composed of only mesenchymal cells and is completely devoid of epithelial elements. Mitoses are present as in Wilms' tumor. Its high cellularity and mitoses are not a reflection of a malignant neoplastic growth but rather of immaturity of tissue in an immature host.

Most mesoblastic nephromas appear in the immediate neonatal period, rarely beyond the first month or two of life. They make up 50% of all renal tumors in the first year of life and an even higher proportion of those appearing in the first days of life. In older children, the tumor attains a large size although the same histologic features as those seen in the neonate persist. Some of the cases show aggressive potential, but dissemination has not been reported.

Although the tumor can be pathologically distinguished from Wilms' tumor, the roentgenographic studies, including excretory urography and angiography, cannot make this distinction.[14] Radiological examination is of great value in determining the size of a presenting mass and in assessing its tissue of origin and its basic nature, namely, solid, cystic or obstructive. Plain film radiography of the abdomen is useful in demonstrating calcifications and bowel displacement. Excretory urography aids in differentiating renal and extra-renal origin of the abdominal mass and at times may differentiate cystic from solid lesions. The latter discrimination may also be augmented by ultrasonography. Renal tumors of the newborn and infants usually show distorted and displaced calyces in the uninvolved part of the kidney similar to the urogram seen in Wilms' tumors in older patients. Angiography offers nothing specific to indicate the benign nature of the mass; the intra-renal arteries usually show the localized changes noted in Wilms' tumor.[14 16]

PROGNOSIS AND TREATMENT

From the cases documented in the literature, as well as our own, survival is excellent following nephrectomy. The cases of reported deaths were due to overzealous treatment of the disease. If the tumor is not completely excised, local recurrence can occur. The appropriate treatment is surgical excision alone inasmuch as the tumor does not respond to chemotherapy or radiation therapy.

SUMMARY

1. Most congenital renal tumors are the benign congenital mesoblastic nephroma of infancy and should be differentiated from the malignant Wilms' tumor, from which it can be pathologically distinguished but with which, in the past, it has been clinically and pathologically confused.

2. The use of the terms, neonatal Wilms' tumor and benign Wilms' tumor, to describe this entity should be avoided and discouraged.

3. Although radiologic studies including plain film roentgenography, excretory urography, angiography, and ultrasonography may be of value in localization of an abdominal mass to a renal origin and may provide information regarding its basic nature, radiologic differentiation of congenital mesoblastic nephroma from Wilms' tumor is usually not possible.

4. Prompt nephrectomy is the treatment of choice; chemotherapy and radiation therapy with their potentially harmful effects are to be avoided assiduously.

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PNEUMATOSIS CYSTOIDES INTESTINALIS ASSOCIATED WITH JEJUNOILEO BYPASS

A Case Report

LYLE H. BOYEA, M.D.
JORGE O'LEARY, M.D.

An interesting documentation of a rare complication in a procedure that still presents so many alterations in metabolism and physiology that its indications remain debatable.

Recently a case of pneumatosis intestinalis complicating jejunoileo bypass was reported by Martyak and Curtis[1] as the first reported case of this complication following jejunoileo bypass. In the case described by these authors, the pneumatosis intestinalis involved only the bypassed segment of small intestines. That patient had a similar procedure to the case described in this article. The case herein presented had some interesting aspects in and above the presence of the pneumatosis intestinalis in that the patient presented with a complication of the pneumatosis intestinalis and surgery was carried out because of this complication.

Pneumatosis cystoides intestinalis remains a condition of relative rarity and of obscure etiology. It has been reported in association with numerous other systemic conditions,[2] as well as gastrointestinal conditions, but never in such a consistent fashion as to suggest a particular etiological relationship. The above authors suggested the possibility of disturbed pressure relationships at the ileo-colonic anastomosis as possibly being etiologically related to the condition. In any event, it would seem with the publication of the above report and the current report that the association may be somewhat more than coincidental and might be logically looked for and anticipated in persons living with this iatrogenic malfunction of the intestinal tract.

Submitted for publication 9 76.

From: Morenci Hospital, Morenci, Arizona, 85540

CASE REPORT

The patient is a 44 year old female who was admitted to the Morenci Hospital on 1-3-76 because of abdominal pain. The pain primarily was in the right upper quadrant of the abdomen, associated with nausea and vomiting. It had been present for two days and had been increasing in severity. There had been no fever or jaundice. There had been no change in bowel habits and no urinary symptoms and no respiratory symptoms.

Past history is quite significant. Although she had had minor illnesses in the past, her only significant chronic difficulty had been obesity. Various attempts at weight reduction had all failed. Finally in June of 1974, at a weight of 312 pounds, she was considered for jejunoileo bypass. Preoperative studies noted a moderate hypertension, generally in the range of 170 100, and a moderately elevated blood uric acid. No other abnormalities were noted upon further physical examination or chemical screening. On 6-5-74 she underwent a jejunoileo bypass elsewhere. At that time a liver biopsy was essentially normal. No evidence of cholelithiasis was noted. No abnormalities of the small intestine were noted, grossly, and an ileal biopsy was reported as normal. The jejunoileo anastomosis was created 12 inches below the ligament of Treitz to 9 inches of distal ileum. The isolated segment was treated by closing the proximal end and tacking to the proximal jejunum. The distal end of the isolated segment was treated by end-to-side anastomosis to the sigmoid colon. This procedure was tolerated well and the patient started to lose weight promptly. The postoperative course was, however, complicated by numerous symptoms which were concurrent with her progressive weight loss. The weight went from 310 pounds to 160 pounds at the time of this admission. However, meanwhile she was plagued intermittently with diarrhea, which never became dangerous in amount although somewhat of a nuisance and was variable in its intensity. This was moderately well controlled with symptomatic drugs. She was also bothered by numbness, tingling, weakness, and was repeatedly noted to have serum calcium in the range of 5-6 milligrams, consistently low serum magnesiums in the range of .5 to .7 milligrams per cent. Serum potassiums were commonly in the 2.5 to 3 mEq. per liter range. She developed no evidence of jaundice. SGOT remained within normal levels, as did the alkaline phosphatase, and blood glucose remained within normal levels. The above symptoms were fairly well controlled by the use of magnesium sulphate supplement, by vitamin D supplement, and by B-12 supplement. During the few months prior to admission she also had some intermittent vague abdominal pain, usually in the right upper quadrant, occasionally associated with nausea, and a cholecystogram done in November of 1975 suggested the presence of a single, sizeable biliary calculus.

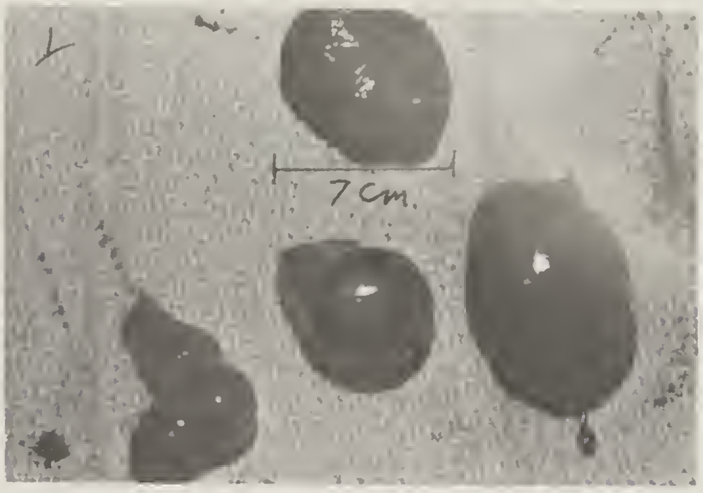

Figure 1

Excised air cysts, including the offending hemorrhagic cysts.

Physical examination upon admission to the hospital revealed her to be well-nourished, well-developed, with evidence of weight loss. Chvostek and Trousseau signs were negative. There was fairly well localized tenderness in the right upper quadrant of the abdomen near the midline, without apparent mass. The old abdominal wounds were well healed. Laboratory studies at this admission included a repeat oral cholecystogram, revealing a non-opaque gallstone. Upper G.I. series was felt to be normal, with a normally functioning ileojujunal anastomosis. Complete blood count was normal. Urinalysis was normal. The serum potassium was 2.8 chlorides 100, sodium 140, serum calcium 8.7, prothrombin time was normal, alkaline phosphatase, serum bilirubin, and SGOT were normal.

Following the above studies, a laparotomy was carried out through a right subcostal incision with the intention of performing a cholecystectomy. The findings of the operation were of an uninflamed gallbladder with a single 1 cm. calculus. However, the striking finding was involvement of the isolated segment of the small intestine throughout its length with typical pneumatosis cystoides intestinalis and in the

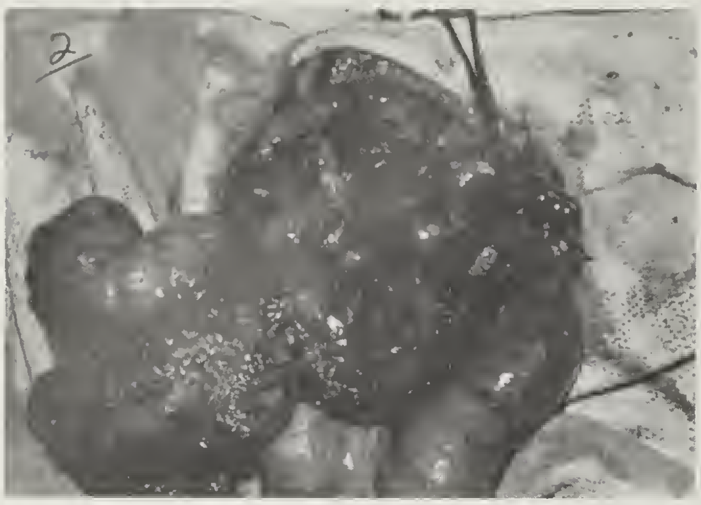

Figure 2

General view of the multiple air cysts involving the isolated segment of small intestine.

cephalad portion of the isolated segment several large cysts were present measuring more than 2 inches in diameter and one of these was filled with blood, apparently from recent hemorrhage. It was felt that this hemorrhagic cyst was the actual source of the patient's presenting acute symptoms rather than the uninflamed gallbladder.

At surgery a cholecystectomy was performed and the hemorrhagic cyst and several other of the larger cysts were excised. They were easily removed from the serosa of the jejunum with no apparent communication with the lumen being detectable. The jejunoileo bypass was left intact. The postoperative course was satisfactory in all respects. She was dismissed from the hospital on the 6th postoperative day without apparent complication.

The patient has been followed for a subsequent 8 months, continuing to have modest intermittent diarrhea but with no recurrence of severe abdominal discomfort to date. She continues on the usual supplements mentioned above. Her weight is holding steady between 160 and 175 pounds.

Interestingly in this particular case, the pneumatosis intestinalis could not be diagnosed on plain films or barium contrast films even in retrospect.

The rather rapid development of cholelithiasis following the institution of the bypass may also be of some interest.

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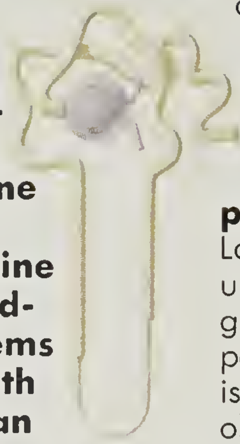


The Lasix® site of action is a distinctive advantage—for it results in fewer electrolytes in each liter of urine than with other commonly used diuretics.[1,2] That is why, if you control urine volume through individual dosage adjustment, you may face fewer problems with electrolyte disturbances.[3] As with any diuretic, electrolyte depletion can occur during therapy with furosemide. Careful clinical and laboratory observations are advisable.

The primary site of Lasix® action—at the loop of Henle—distinguishes Lasix from other commonly used diuretics. The action at this site enables Lasix to produce a dilute urine,[4] which may be the basis for its relative freedom from electrolyte disturbances (hypokalemia).[3] When the edematous patient has been stabilized, excessive diuresis can usually be avoided by simple dosage adjustment. And, through regulation of urine volume by this dosag adjustment, you can control electrolyte excretio Careful medical supervision is required.

Wide dose-related effectiveness for precise control of diuretic response. The Lasix® effect is dose-related. A prompt diuresis usually ensues with a single dose of 20 to 80 mg, given preferably in the morning. Depending on th patient's response, a second dose may be administered 6 to 8 hours later. Clinical and laboratory observations are advisable when doses exceeding 80 mg/day are given for prolonged periods. Whe maintenance of the edema-free state is desired, dosage may be adjusted to as low as 20 mg.

(Please see the dosage and administration section on the last page of this advertisement.)

Highly effective/short acting. The effectiveness of Lasix® has been clearly establishe in hundreds of thousands of patients in a decade o clinical experience. Consequently, Lasix is the most selected diuretic agent today in the treatme of edemas associated with congestive heart failur

because fewer electrolytes
get into each liter of
urine than with other commonly
used diuretics...

LASIX® (FUROSEMIDE)

the diuretic for both initial & maintenance therapy in cardiac edema

The onset of diuresis usually occurs within an
our and is usually complete within 6 to 8 hours;
us, patient inconvenience is minimized.

Useful in patients with renal impair-
ent and severely depressed renal func-
on. Unlike other commonly used diuretics, Lasix®
n be useful in patients with renal impairment
d severely depressed renal function. Even in the
esence of renal insufficiency with depressed
omerular filtration, acidosis, alkalosis or hypo-
buminemia, Lasix has been found effective and
ell tolerated.[5] If increasing azotemia and oliguria
cur during treatment of severe progressive renal
sease, the drug should be discontinued.

(See next page for prescribing information.)

References: 1. Peltola, P.: Furosemide (Lasix®) as a diuretic, *Acta Medica Scandinavica, 177*:6, 1965. 2. Mudge, G.H., in Goodman, L.S. and Gilman, A. (eds.), The Pharmacological Basis of Therapeutics, 4th ed., 1970, p. 852. 3. Manner, R.J., Brechbill, D.O., and DeWitt, Karen: Prevalence of hypokalemia in diuretic therapy, *Clin. Med.* 79:19, November 1972. 4. Schrier, R.W., *et al.*: Effect of furosemide on free water excretion in edematous patients with hyponatremia, *Kidney Int.* 3:30, 1973. 5. Muth, R.G.: Diuretic response to furosemide in the presence of renal insufficiency, *J.A.M.A. 195*:1066, March 1966.

the basic unit

LASIX® 40 mg tablets

(FUROSEMIDE)

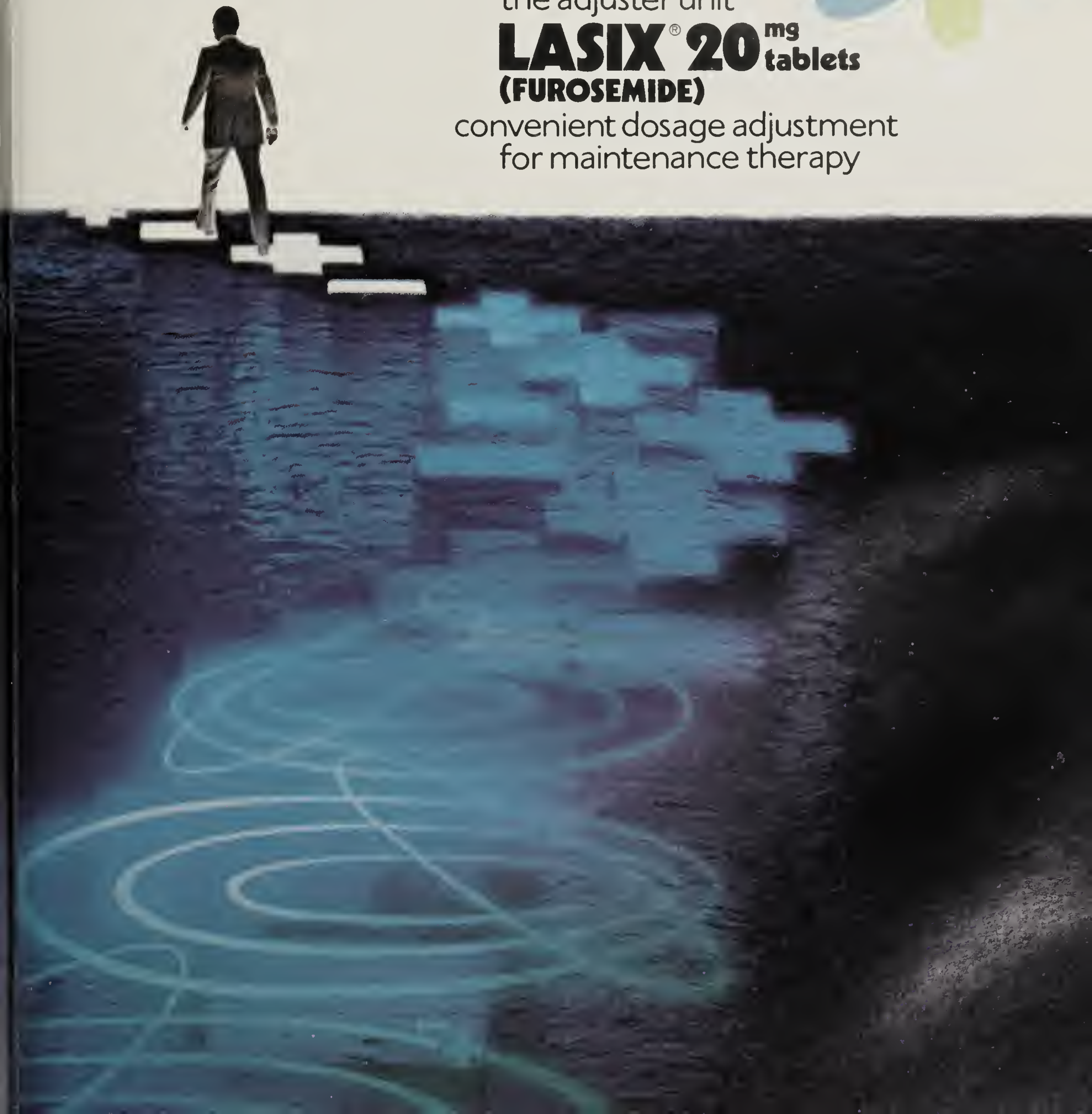
unsurpassed effectiveness
for prompt reduction
of edema

the adjuster unit

LASIX® 20 mg tablets

(FUROSEMIDE)

convenient dosage adjustment
for maintenance therapy



LASIX® 40 mg and 20 mg tablets. (FUROSEMIDE)

logical long-term diuretic therapy for cardiac edema

A brief summary of the Prescribing Information for

Lasix® (furosemide) Tablets 20 mg and 40 mg

WARNING—Lasix (furosemide) is a potent diuretic which if given in excessive amounts can lead to a profound diuresis with water and electrolyte depletion. Therefore, careful medical supervision is required, and dose and dose schedule have to be adjusted to the individual patient's needs. (See under "Dosage and Administration.")

Indications—Lasix (furosemide) is indicated for the treatment of the edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Hypertension—Lasix (furosemide) may be used for the treatment of hypertension alone or in combination with other antihypertensive drugs. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controllable with Lasix (furosemide) alone.

CONTRAINDICATIONS—Because animal reproductive studies have shown that Lasix (furosemide) may cause fetal abnormalities, the drug is contraindicated in women of childbearing potential. (See "Additional Information.")

Lasix (furosemide) is contraindicated in anuria. If increasing azotemia and oliguria occur during treatment of severe progressive renal disease, the drug should be discontinued. In hepatic coma and in states of electrolyte depletion, therapy should not be instituted until the basic condition is improved or corrected. Lasix (furosemide) is contraindicated in patients with a history of hypersensitivity to this compound.

Warnings—Excessive diuresis may result in dehydration and reduction in blood volume, with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients. Excessive loss of potassium in patients receiving digitalis glycosides may precipitate digitalis toxicity. Care should also be exercised in patients receiving potassium depleting steroids.

Frequent serum electrolyte, CO_2 and BUN determinations should be performed during the first few months of therapy and periodically thereafter, and abnormalities corrected or the drug temporarily withdrawn.

In patients with hepatic cirrhosis and ascites, initiation of therapy with Lasix (furosemide) is best carried out in the hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma; therefore, strict observation is necessary during the period of diuresis. Supplemental potassium chloride and, if required, an aldosterone antagonist are helpful in preventing hypokalemia and metabolic alkalosis.

Patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

In those instances where potassium supplementation is required, an oral liquid preparation should be used rather than enteric-coated potassium salts.

There have been several reports, published and unpublished, concerning nonspecific small-bowel lesions consisting of stenosis, with or without ulceration, associated with the administration of enteric-coated thiazides with potassium salts. These lesions may occur with enteric-coated potassium tablets alone or when they are used with nonenteric-coated thiazides, or certain other oral diuretics.

These small-bowel lesions have caused obstruction, hemorrhage, and perforation. Surgery was frequently required, and deaths have occurred.

Available information tends to implicate enteric-coated potassium salts, although lesions of this type also occur spontaneously. Therefore, coated potassium-containing formulations should be administered only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Patients with known sulfonamide sensitivity may show allergic reactions to Lasix (furosemide).

Precautions—As with any potent diuretic, electrolyte depletion may occur during therapy with Lasix (furosemide), especially in patients receiving higher doses and a restricted salt intake. Electrolyte depletion may manifest itself by weakness, dizziness, lethargy, leg cramps, anorexia, vomiting, and/or mental confusion.

Asymptomatic hyperuricemia can occur and gout may rarely be precipitated. Reversible elevations of BUN may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

When parenteral use of Lasix (furosemide) precedes its oral use, it should be kept in mind that cases of tinnitus and reversible hearing impairment have been reported. There have also been some reports of cases in which irreversible hearing impairment occurred. Usually, ototoxicity has been reported when Lasix (furosemide) was injected rapidly in patients with severe impairment of renal function at doses exceeding several times the usual recommended dose and in whom other drugs known to be ototoxic were often given. If the physician elects to use high dose parenteral therapy in patients with severely impaired renal function, controlled intravenous infusion is advisable (for adults, it has been reported that an infusion rate not exceeding 4 mg Lasix [furosemide] per minute has been used).

Increases in blood glucose, and alterations in glucose tolerance tests with abnormalities of the fasting and two-hour postprandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Lasix (furosemide) may lower serum calcium levels, and rare cases of tetany have been reported.

Patients receiving high doses of salicylates, in conjunction with Lasix (furosemide) may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Diuretics such as furosemide may enhance the nephrotoxicity of cephaloridine. Therefore, Lasix (furosemide) and cephaloridine should not be administered simultaneously.

Sulfonamide diuretics have been reported to decrease arterial responsiveness to pressor amines and to enhance the effect of tubocurarine. Great caution should be exercised in administering curare or its derivatives to patients undergoing therapy with Lasix (furosemide), and it is advisable to discontinue Lasix (furosemide) for one week prior to any elective surgery.

Adverse Reactions—Various forms of dermatitis, including urticaria and rare forms of exfoliative dermatitis, erythema multiforme, pruritus, paresthesia, blurring of vision, postural hypotension, nausea, vomiting, or diarrhea.

Anemia, leukopenia, aplastic anemia, and thrombocytopenia (with purpura). Rare cases of agranulocytosis which responded to treatment.

In addition, the following rare adverse reactions have been reported; however, relationship to the drug has not been established with certainty: sweet taste, oral and gastric burning, paradoxical swelling, headache, jaundice, thrombophlebitis and emboli and acute pancreatitis.

Lasix (furosemide)-induced diuresis may be accompanied by weakness, fatigue, lightheadedness or dizziness, muscle cramps, thirst, increased perspiration, urinary bladder spasm, and symptoms of urinary frequency.

Dosage and Administration

ADULTS

The usual adult dose of Lasix (furosemide) is 20 to 80 mg given as a single dose.

If the diuretic response with a single dose of 20 to 80 mg is not satisfactory, the following schedule should be used: Increase this dose by increments of 20 or 40 mg not sooner than 6 to 8 hours after the previous dose until the desired diuretic effect has been obtained. This individually determined single dose should then be given once or twice daily. The dose of Lasix (furosemide) may be carefully titrated up to 600 mg per day in those patients with severe clinical edematous states.

With doses exceeding 80 mg/day and given for prolonged periods, careful clinical and laboratory observations are particularly advisable.

Hypertension—The usual dose of Lasix (furosemide) is 40 mg twice daily both for initiation of therapy and for maintenance. Careful observations for changes in blood pressure must be made when this compound is used with other antihypertensive drugs, especially during initial therapy. The dosage of other agents must be reduced by at least 50 percent as soon as Lasix (furosemide) is added to the regimen to prevent excessive drop in blood pressure. As the blood pressure falls under the potentiating effect of Lasix (furosemide), a further reduction in dosage, or even discontinuation, of other antihypertensive drugs may be necessary. It is further recommended, if 40 mg twice daily does not lead to a clinically satisfactory response, to add other hypotensive agents, e.g., reserpine, rather than to increase the dose of Lasix (furosemide).

INFANTS AND CHILDREN

Pediatric Administration: The usual initial dose of oral Lasix in infants and children is 2 mg/kg body weight, given as a single dose. If the diuretic response is not satisfactory after the initial dose, dosage may be increased by 1 or 2 mg/kg not sooner than 6 to 8 hours after the previous dose. Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

How Supplied—Lasix Tablets 40 mg (furosemide) supplied as white, round, monogrammed, scored tablets.

Lasix Tablets 20 mg (furosemide) supplied as white, oval, monogrammed tablets.

Note: Dispense in dark containers. Exposure to light may cause slight discoloration which, however, does not alter potency.

Additional Information

Toxicology

The acute toxicity of Lasix (furosemide) has been determined in mice, rats, and dogs. In all three animal species, the oral LD_{50} of Lasix (furosemide) exceeded 1000 mg/kg of body weight, while the intravenous LD_{50} ranged from 300 to 680 mg/kg. Intragastric injection of the drug in newborn rats resulted in an LD_{50} of 380 mg/kg.

The acute toxicity of high doses of Lasix (furosemide) was characterized by convulsions, paralysis, and collapse. Surviving animals often became dehydrated and depleted of electrolytes due to the diuresis induced by Lasix (furosemide). In the newborn rats, intragastric injection of the drug caused hyperactivity and anorexia.

Chronic toxicity studies with Lasix (furosemide) were done in rats and dogs. In a one-year study in rats, renal tubular degeneration occurred, with all doses higher than 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day). A six-month study in dogs revealed calcification and scarring of the renal parenchyma at all doses above 10 mg/kg (83 percent of the maximal recommended human dose of 600 mg per day).

Reproductive Studies

The effects of Lasix (furosemide) on embryonic and fetal development and on pregnant dams were studied in mice, rats, and rabbits.

Lasix (furosemide) caused unexplained maternal deaths and abortions in the rabbit when 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day) was administered between days 12 to 17 of gestation. In a previous study the lowest dose of only 25 mg/kg (2 times the maximal recommended human dose of 600 mg per day) caused maternal deaths and abortions. In a third study, none of the pregnant rabbits survived a dose of 100 mg/kg. Data from the above studies indicate fetal lethality which can precede maternal deaths.

The results of the mouse study and one of the three rabbit studies also showed an increased incidence of hydronephrosis (distention of the renal pelvis and, in some cases, of the ureters) in fetuses derived from treated dams as compared to the incidence in fetuses from the control group.

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TO BARE OR NOT TO BARE, THAT'S THE QUESTION

JOHN W. KENNEDY, M.D.

The American Medical Association in a recent poll found that one out of eight physicians responding are practicing without malpractice insurance. This poll also revealed that one out of three of those responding were considering dropping their insurance. Most gave as the cause the escalating cost of the insurance premiums and of the Pacific states 51% of the physicians who responded, were considering "going bare".

On page 1045 Dr. Howard Holmes of Eloy, describes the problem that confronts him — "going bare" — in an Arizona agricultural community. The rising cost of malpractice insurance and office overhead has already forced a number of senior practitioners, in both urban and rural areas, nation-wide, to shut up shop. What a waste of mature medical talent.

It remains to be seen whether the new programs in family and primary medicine will be sufficient to supply the need in both the isolated communities and the urban areas.

The plight of another Arizona physician, denied hospital privileges, when he went "bare", Dr. Wallace Reed of Phoenix, has recently been described in detail.[1] Dr. Reed was immediately suspended from the medical staff of two of the major Phoenix hospitals, two other hospitals did not suspend him. "Dr. Reed in asking these two hospitals to lift his suspension called the following to their attention:

"The criteria for medical staff privileges are embodied in Standard VII of the 'Governing Body and Management' Section of the Accreditation Manual for Hospitals, Joint Commission on Accreditation of Hospitals. This well recognized work on practical ethics of physician and hospital relationship states:

"The governing body shall delegate to the Medical Staff the authority to evaluate the professional competence of the staff members and applicants of staff privileges; it shall hold the Medical Staff responsible for making recommendations to the governing body concerning initial staff appointments, reappointments, and the assignment or curtailment of privileges.

"No applicant shall be denied Medical Staff membership and or clinical privileges on the basis of sex, race, creed, color or national origin, or on the basis of any other criterion lacking professional justification".[1]

Hospital and physician relationship, in recent years, has become more divisive. But as many in both professions acknowledge this is ill considered and does not contribute to the best of patient care. After all, that is the only excuse for physicians or hospitals and all of the ancillary personnel and institutions. Patient care, seems to be frequently lost sight of in the heat of the fray.

We are unfortunately subject to the same human frailty as all other professions and activities on this planet. We react to crises when they are crushing us but we fail to anticipate them. The malpractice crisis was indeed such an event, as long as seven years ago there were physicians in this community and State who were either denied renewal of their liability insurance policy or were "rated upward". In some instances there indeed may have been justification but in others it was simply because the doctor had either been threatened with a suit, or a suit had been filed and brought to court and the physician exonerated. Nonetheless organized medicine in this State took no more than a passing interest, we stuck our head deep in the proverbial sand and whispered to ourselves, "it won't happen to me".

Editor's Page

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So the malpractice crisis committee was formed on a State level and did a very creditable piece of work, under trying circumstances. No one can safely say that this is a long term solution. Indeed there has been more than a little discussion that a constitutional amendment, in the State, would be necessary to finally bring it satisfactorily under control. This will take the undivided effort and substance of every physician in the State of Arizona.

Now we are provided with another possible alternative, going "bare", but this also demands the attention and even more dollars to supply the legal force necessary to obtain a court decision . The courts will apparently have to establish whether or not the hospital governing board has the power, without the acquiescence of the medical staff, to force every physician who has privileges in their institution to acquire and keep in force liability insurance, no matter what the cost.

Indeed the struggle of these "going bare" physicians for their financial and professional survival may "be the fight of all other physicians".

Reference: (1) "Going Bare in Phoenix". Private Practice, October 1976.

Dean's Page

NUCLEAR MEDICINE

NEAL A. VANSELOW, M.D.

Nuclear Medicine is one of the newest of the recognized medical specialties. The first Board Examination in Nuclear Medicine was given in 1972. Unlike most other medical specialties, Nuclear Medicine does not have its roots in antiquity, but dates back to the beginnings of the radioactive tracer technique, which reached its maturity after the second World War.

Nuclear Medicine has developed into a field that deals primarily with diagnostic imaging. Because the distribution of radioactive tracer in the body requires that certain body functions be intact, Nuclear Medicine images are functional images in that they depict a certain body function (e.g., thyroid uptake, reticuloendothelial function, intact blood-brain barrier, etc.). Nuclear Medicine studies, for the most part, carry virtually no risk to the patient and most studies can be done on patients of any age.

Nuclear Medicine images differ from conventional radiographic images in that they are images of some body function or other. For example, while an X-ray of a hand would look the same whether the patient were alive or not, a bone scan requires that the bone actively take up the radioactive isotope; in this sense the bone scan reflects the "livingness" of the bone. If the organ being studied has lost its function there will be no image.

In addition to the standard diagnostic tests of body function that have been available for several years, some newer developments have extended the scope of Nuclear Medicine into new areas. Newer agents for bone scanning deliver a much lower radiation dose than agents previously used, and bone scans can now be done safely on children and on patients without malignant disease. This has opened up bone scanning for the evaluation of patients with orthopedic and other benign bone disease. Bone scans are often helpful in the early diagnosis of fractures, osteomyelitis, Paget's disease, and other conditions in which radiographic studies may be inconclusive.

Several techniques have been developed over the past few years for the diagnosis of heart disease, including the development of technetium 99m pyrophosphate studies for myocardial infarction, thallium 201 studies of myocardial perfusion, and measurements of cardiac ejection. Pyrophosphate studies of the heart for myocardial infarction are quite sensitive and detect myocardial ischemic disease between 12 hours and 7 days of the ischemic event. The mechanism of action seems to be that damaged myocardium accumulates calcium; the labeled (radioactive) pyrophosphate combines with the calcium and accumulates with it in the injured heart muscle. These diagnostic tests are being done in the cardiac intensive care unit to establish the existence of recent myocardial infarction in patients who have a history of old MI or whose history otherwise complicates the diagnosis. Tumor-seeking radiopharmaceuticals have been one of the main goals of Nuclear Medicine since its inception. Recently bleomycin, a chemotherapeutic agent, has been labeled with indium 111 or with cobalt 57. In selective cases this radiopharmaceutical has been an effective aid in delineating tumor that otherwise would have been impossible to image by any other technique.

Blood flow through an organ can be determined quantitatively by measuring the washout of a diffusible tracer. This generally requires a computer and some special approaches, but the tracer technique is virtually free from hazards. Xenon-133, a radioactive gas dissolved in saline, is injected via catheter into an organ during conventional angiography. The tracer diffuses through the tissues and is washed out by subsequent blood flow. The rate of washout is an indication of the blood flow.

Although Nuclear Medicine techniques are essentially free from hazard, our concern for radiation safety requires that all new diagnostic procedures be thoroughly validated in hospitals equipped to perform such investigations. As soon as the safety and effectiveness of these diagnostic procedures have been validated, the procedures are made available to all physicians for routine use. As the development of this exciting new field continues, we should expect to see more sensitive and more specific agents for the detection and staging of a wide variety of disorders.

Noel A. Vanselow

President's Page

FAVORABLE SITUATIONS

EDWARD SATTENSPIEL, M.D.

Some favorable situations have developed for us, both on the national and local scene, believe it or not!

The Health Professions Education Assistance Act of 1976 has been signed by the president and is now law. This legislation was the subject of intense debate in both the Senate and House and in Conference Committee. Because of strong objections raised by the medical profession through the A.M.A. and others, some of the most objectionable provisions were deleted in the final bill which passed. The requirement that medical schools set aside a large percentage of their enrollment for National Health Service Corps scholarships was deleted, along with required service in physician shortage areas. The federal program for creating national standards for model state laws for licensure and relicensure of physicians was removed from the act. Provisions aimed at effecting affiliation of all hospital residency programs with a medical school were thrown out. Granting the need for federal financial aid to medical schools and medical students, we can live with this new legislation at least.

On the local scene, I've been very impressed with the efforts of Ar.M.A.'s underwriting committee. This committee provides the Mutual Insurance Co. of Arizona with regular professional advice and assistance for properly underwriting physicians and other health care providers. Although committee meetings are long and arduous, and their decisions cannot always please everyone, they do provide a sincere and objective group of peers for Arizona physicians to deal with, rather than some impersonal corporate entity in Connecticut or California.

Our monthly TV program "Medical Opinion" moderated by Dr. Duval on KOOL-TV for the past 32 months has been the most watched program during its time slot. It also had more viewers than any of the other three programs KOOL aired on a weekly tandem basis during that 6:30 to 7:00 P.M. Saturday time slot. Because of this excellent public reaction, KOOL-TV has requested and we have agreed to do this program on a weekly basis from 5:00 to 5:30 P.M. each Saturday. Dr. Duval has agreed to continue as moderator for approximately half of the programs. KOOL staff member, Dennis Johnson, who has handled virtually all of the station's medically-related news coverage for several years, would serve as moderator on alternate weeks. Ar.M.A. through its Public Relations Committee under the chairmanship of Dr. Charles Finney, will be responsible for selecting topics, providing the station with background information on each, and for recruiting the two physician panelists for each program. Many of you may be asked to volunteer. This is an outstanding education and image-improving activity, which we trust will continue to thrive.

Clinical Oncology
In Arizona

RADIOTHERAPY IN THE MANAGEMENT OF THE LEUKEMIAS

ROBERT HEUSINKVELD, M.D., Ph.D.

Editors: Stephen E. Jones, M.D., Associate Professor of Medicine, Section of Hematology and Oncology, University of Arizona College of Medicine, Tucson; Arizona 85724; Robert H. Thoeny, M.D., Dir., Radiation Oncology, Good Samaritan Hospital, Phoenix, Arizona 85006: Darwin W. Neubauer, M.D., General Surgeon, 720 Country Club Road, Tucson, 85716.

INTRODUCTION

The extreme sensitivity of leukemic cells to ionizing radiation was recognized as early as 1902 when X-rays were first used to treat a patient with leukemia, and until the advent of modern chemotherapy in the 1950's ionizing radiation was the mainstay of leukemia treatment. Today, palliative irradiation is often used to relieve the discomfort of patients with symptomatic extramedullary leukemic infiltrates. In addition, there are a few specific situations in which radiotherapy may be used in the primary treatment of leukemias. The purpose of this article is to review the ways in which radiotherapy contributes to the management of patients with leukemia.

Chronic Leukemias

Chronic Myelogenous Leukemia (CML): Splenic irradiation was the treatment of choice for patients with CML before the advent of chemotherapy. However, comparative trials have demonstrated that busulfan is more effective in controlling CML,[1] [2] and today the major role of radiotherapy in the treatment of CML is in the palliation of symptomatic infiltrates which appear during the accelerated or terminal phase of the disease. Painful splenomegaly can almost always be alleviated by irradiation, although there is the risk of severe pancytopenia, particularly in patients with poor bone marrow function. Splenic irradiation should be initiated with caution, starting with doses as low as 50 rads on alternate days for thrombocytopenic patients or for those receiving intensive chemotherapy. A useful but not universally appreciated fact is that the bone and joint pains which frequently afflict patients with terminal CML can be relieved with single small doses of radiation. Multiple sites can be treated in a single session, eliminating a major source of discomfort.

From: Division of Radiation Oncology, U of A Health Sciences Center, Tucson, AZ 85721

Chronic Lymphocytic Leukemia (CLL): Lymphocytes are particularly radiosensitive, and a low dose of radiation will eliminate the painful or cumbersome lymphadenopathy and symptomatic extranodal infiltrates of CLL. In addition, systemic treatment of CLL with radiation has been attempted in the form of splenic irradiation, thymic irradiation, extracorporeal irradiation of blood (ECIB), administration of radioactive phosphorus (^{32}P), and whole-body X or gamma irradiation (WBI) with generally encouraging results.

Splenic irradiation, in addition to providing relief of pain and reducing spleen size, is frequently associated with a general amelioration of CLL. A rise in hematocrit and platelet count and a reduction of peripheral lymphadenopathy are sometimes observed following splenic irradiation.[3] Although the exact mechanism of this "abscopal" effect of splenic irradiation is poorly understood, pre- and post-irradiation kinetic studies in patients with CLL have demonstrated both decreased splenic hemolysis and increased normal hematopoiesis.[4] It seems probable that reduction of splenomegaly in patients with CLL reduces the hypersplenism which may be associated with splenomegaly of any etiology, thus restoring hematocrit and platelet counts towards normal. Also, since the spleen is a major repository of leukemic lymphocytes in patients with CLL and may be a major site of lymphocyte production,[5] splenic irradiation has the additional advantage of destroying a large portion of the body burden of abnormal lymphocytes without damaging hematopoietic sites. Other interactions between the spleen and bone marrow have long been suspected, and these may also influence the favorable effect of splenic irradiation on hematopoiesis. The precipitous thrombocytopenia noted with splenic irradiation in CML is not commonly seen with splenic irradiation in CLL.

Irradiation of the thymus has been reported to reduce peripheral lymphocytosis and infiltration of lymph nodes, liver, spleen, and bone marrow in patients with CLL as well as or perhaps more effectively than chlorambucil.[6] This novel approach to the treatment of CLL has important pathophysiologic as well as therapeutic implications, but it requires further evaluation. Thymic irradiation, like splenic irradiation, has the advantage of sparing the bone marrow, and it will be of great interest to see how frequently this form of treatment can benefit patients with CLL, especially those with the bone marrow failure so frequently encountered during the advanced stages of disease.

Extracorporeal irradiation of blood (ECIB) has been tried in an attempt to destroy leukemic lymphocytes without suppressing the bone marrow. ECIB is one of the few therapeutic maneuvers available for the treatment of advanced CLL when bone marrow reserve has become inadequate to tolerate further suppression by either chemotherapy or systemic irradiation. In pilot studies, treatment of advanced CLL with ECIB has been shown to be feasible and in some cases beneficial.[7, 8] Marked reduction of lymphocytosis occurs in all patients, and in some patients, there is significant depopulation of extravascular lymphocyte deposits, evidenced by shrinkage of the spleen and enlarged nodes and improvement in hematopoiesis. Unfortunately, a single course of ECIB seldom provides long-term palliation, and the inconvenience of the method has discouraged the repeated use required to provide prolonged benefit. It is possible that use of splenic irradiation together with ECIB might be more effective and still marrow sparing since there appears to be substantial splenic pooling of lymphocytes in patients with CLL.

Radioactive phosphorus (^{32}P) has been extensively evaluated as a treatment for CLL. Osgood[9] evaluated the effectiveness of intermittent total body irradiation with either x-rays or intravenously administered ^{32}P in a consecutive series of 326 patients with chronic leukemia (212 with CLL and 114 with CML). The median survival from the time of the beginning of treatment in the subgroup of 138 previously untreated CLL patients in this series was 48 months, with 18 patients alive more than 10 years after the initiation of treatment and 25 alive a minimum of 7.5 years after the beginning of treatment. These results, equaled in few contemporary series, were often quoted to support Osgood's theories regarding the effects of irradiation as opposed to chemotherapy on the kinetics of malignant cell proliferation. ^{32}P had no apparent advantage over "equivalent" doses of total body x-irradiation, the mode of irradiation currently arousing the most interest as a method of treating CLL.

Interest in *whole-body X or gamma irradiation (WBI)* as a treatment for CLL has been rekindled by the finding that chemotherapy has not measurably influenced the prognosis of CLL.[10] The most recent experience with WBI of CLL has been reported by Johnson[11] who administers total doses of 100 to 400 rads in small fractions at a frequency adjusted to avoid dangerous myelosuppression. Forty-two patients with progressive disease treated in this manner have had a median actuarial survival of 57 months from initiation of treatment; 34% of these patients have had complete responses (defined as a return to nearly normal blood counts, absence of absolute lymphocytosis or palpable lymph nodes, and less than 30% marrow lymphocytes). Immunoglobulin levels have also returned to normal in most of these complete responders, and the incidence of infections in this group has been significantly lower than in patients with less than complete responses. Survival of the complete responders is significantly longer than that of partial responders. These results are encouraging and suggest that the course of the disease may be reversed in some patients, with possible prolongation of life. However, the possibility that these "complete responders" represent a subgroup of patients with a more favorable prognosis (regardless of the nature of their treatment) cannot be excluded in this nonrandomized series. Clinical trials are under way to clarify this point. At present, WBI has proven to be at least as effective as any other form of treatment of CLL and relatively free of morbidity.

Acute Leukemias

Acute Lymphocytic Leukemia (ALL): ALL is primarily a disease of childhood. Although a high percentage of children with ALL now enter complete and long-lasting remissions following induction chemotherapy, as many as 60% will relapse primarily in the central nervous system (CNS) if treated only with chemotherapy. (Apparently, most chemotherapeutic agents do not reach leukemic stem cells which may be sequestered within the CNS.) Uncontrolled CNS relapses are invariably followed by peripheral dissemination of the leukemia. To eliminate leukemic stem cells in the CNS beyond the reach of systemic chemotherapy, various combinations of CNS irradiation and or intrathecal chemotherapy have been tried. Currently, the safest and most effective regimen appears to be a combination of cranial irradiation and intrathecal administration of methotrexate. This regimen has been reported to completely prevent primary CNS relapse in children treated during their initial continuous complete remission.[12] The combination of cranial irradiation and intrathecal methotrexate treatment is more tolerable than cranio-spinal irradiation in which the entire CNS is treated. In addition, cranio-spinal irradiation may depopulate substantial portions of the bone marrow and compromise programs of maintenance chemotherapy. The complication rate of combined cranial irradiation and intrathecal methotrexate treatment is low, although occasionally progressive leukoencephalopathy will develop. This syndrome has been reported with intrathecal methotrexate and CNS irradiation alone and in combination.

Acute Myelocytic Leukemia (AML): AML is more common in adults than in children. Patients with AML have occasional CNS infiltration and occasionally relapse primarily in the CNS. Primary CNS relapse may be somewhat more common in children than in adults and at some cancer treatment centers, children with AML are given prophylactic treatment to the CNS as in the treatment of ALL. Because involvement of the CNS in adult patients with AML is relatively uncommon and less than 10% of adult patients with AML relapse primarily in the CNS, prophylactic irradiation of the CNS in adults is not routinely practiced.

SUMMARY

The specific uses of radiotherapy in patients with leukemia have been reviewed in this article. In addition, it is important to emphasize that symptoms due to leukemic infiltrates in almost any site inaccessible to or resistant to chemotherapy can usually be relieved by irradiation, regardless of the type of the leukemia.

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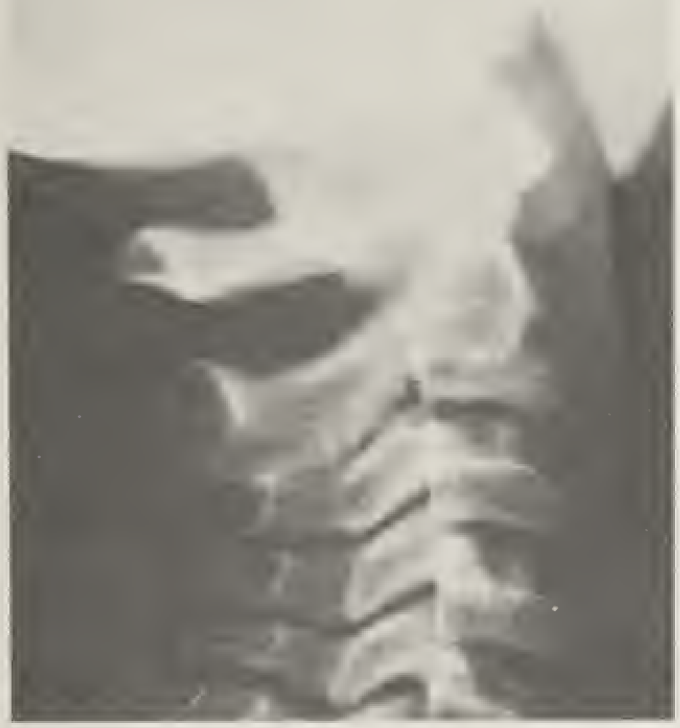

Figure 1

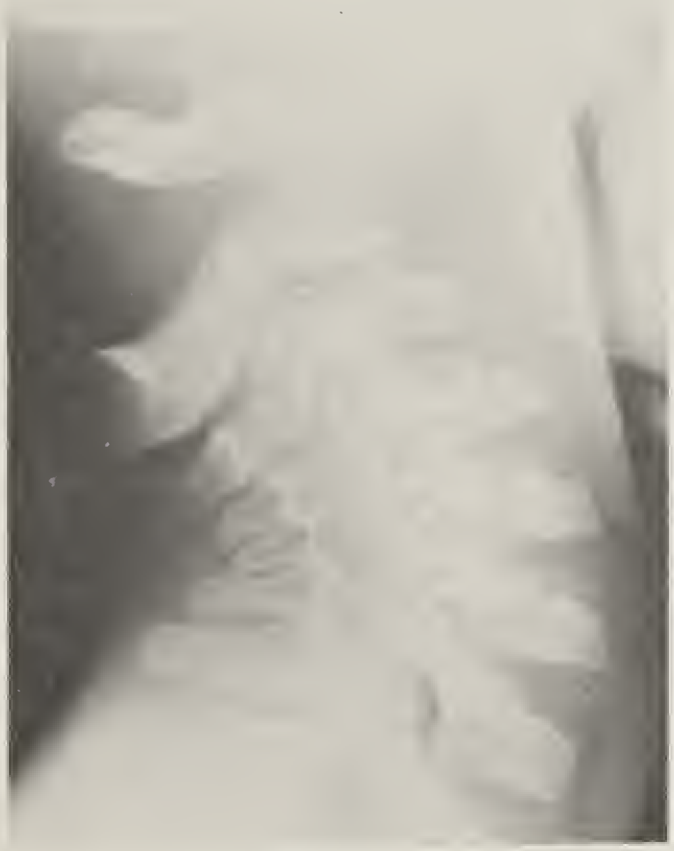

Figure 2

CASE NO. 17

PHILIP STRATEMEIER, M.D.

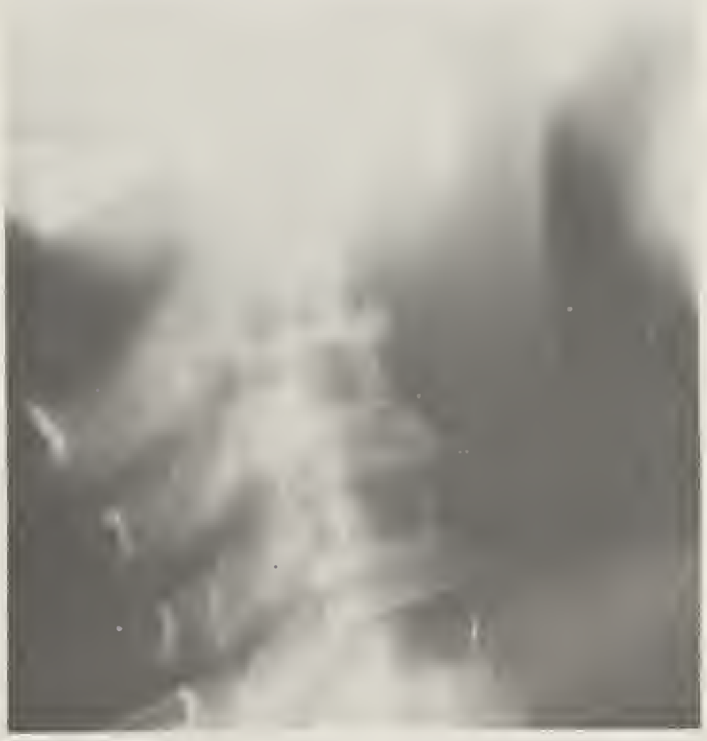

Figure 3

Figure 1 shows a lateral projection of the cervical spine of a 7 year old white male, who had broken his arm and was complaining of neck pain. He returned to the emergency room 3 months later complaining of increased neck pain that had not subsided since his earlier complaint. Figure 2 shows a lateral cervical spine radiograph at that time. Figure 3 is a tomogram obtained shortly after Figure 2. What is your diagnosis?

From: Dept. of Radiology, U of A Health Sciences Center, 85724.

DIAGNOSIS: EOSINOPHILIC GRANULOMA

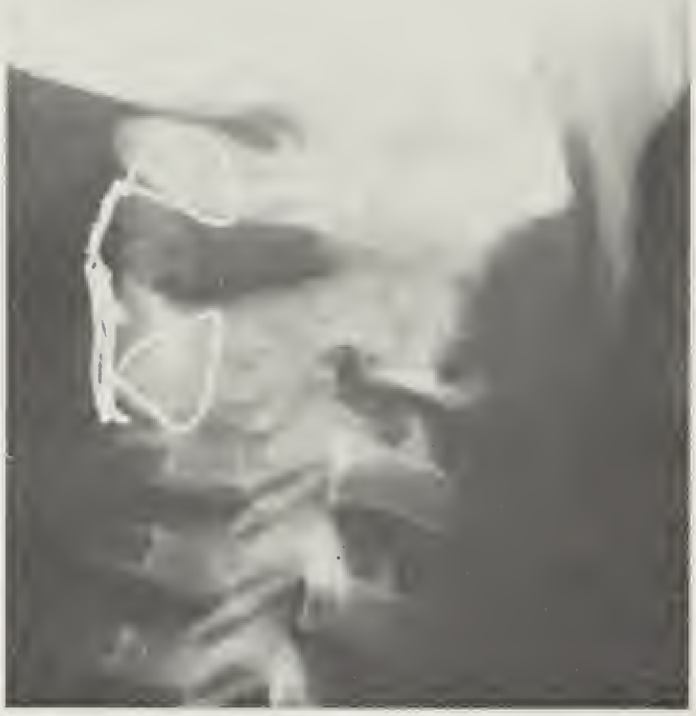

Figure 4

The first radiograph shows a lytic process in the body of C2, though it was not recognized at the time. In the second series, obvious C2 vertebral body destruction is present, including the odontoid process, with subluxation of C1 on C2. No nerve root or cord compression signs were present. The patient was placed in traction and the tomogram of C2 was made to determine the extent of the destruction and to help in planning future therapy. After several weeks in traction and irradiation to the body of C2, the posterior elements of C1 and C2 were stabilized by wires and bone chips (Figure 4).

Eosinophilic granuloma is the least aggressive of the three proliferative disorders that have been collectively termed Histiocytosis X. The other two disorders are Letterer-Siwe and Hand-Schuller-Christian. The three disorders consist of histiocytic proliferation of unknown etiology. There is an associated eosinophilia as well as lymphocytes, and plasma cells, which can make differentiation from reticulum cell sarcoma difficult.

In Letterer-Siwe, the disease is frequently evident before age 3, with fever, otitis media, anemia, hepatosplenomegaly, lymphadenopathy and multiple lytic bone lesions. It is usually fatal. Hand-Schuller-Christian has its onset usually later than Letterer-Siwe. It is characterized by multiple skull defects, exophthalmus, and diabetes insipidus. It is frequently fatal, though patients may live to adult life before death.

Eosinophilia granuloma is also a disease of infancy and childhood. Its most frequent presentation is that of a solitary bone lesion. If multiple lesions are present, then the prognosis is poorer, although an older age at onset is associated with a better survival.

Clinically the most common initial complaint is of local pain and tenderness, frequently with swelling, particularly in the skull. Bone changes occur within six weeks of onset of symptoms. The radiographic appearance in long bones is usually of a solitary lesion, round or ovoid, with a narrow zone of transition. It is most common to the diaphysis and less often to the metaphysis, consisting mostly of medullary destruction with cortical erosion. A periosteal reaction may be present, which is layered, simulating osteomyelitis or Ewing's Sarcoma. In the skull, the appearance is that of a round or oval lytic defect, often multiple, with a "beveled edge" due to diploic expansion into the inner and outer tables at different rates. There is no sclerosis unless the lesion has been treated. In the mandible, the teeth appear to be "floating" in a lucent zone. The vertebral bodies may show destruction or complete collapse (vertebra plana), with the disc space normal or slightly wider.

Differential diagnosis includes trauma, Gaucher's disease, Hodgkin's, leukemia, neurofibromatosis, and hemangiomas. Treatment is by curettage for accesible unifocal lesions as in long bones. Radiotherapy using low doses (less than 1200 rads) has been effective, sometimes combined with corticosteroids in disseminated disease. The vertebral body may be completely reconsitituted. In this patient, surgery and supravoltage therapy were used. After one year there was some sclerosis, but no evidence for regeneration of the destroyed vertebral body.

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Obstetrics and Gynecology

THE ARIZONA MATERNAL MORTALITY COMMITTEE

WILLIAM A. SUSONG, M.D.

DONALD J. ZIEHM, M.D., EDITOR

The Arizona Maternal Mortality Committee was originally established in May, 1965 as a subcommittee of the Perinatal Mortality Committee of the Arizona Medical Association. Dr. Hermann Rhu of Tucson, chairman of the Perinatal Mortality Committee, then appointed Dr. David Pent as chairman of the Maternal Mortality subcommittee. Dr. Pent held this position until 1976. He initially researched the organization and function of other maternal mortality committees throughout the country and developed the forms and letters that were needed to identify and investigate maternal deaths in the state. With this background the committee began a study of maternal deaths as they occured from July, 1966.

A maternal death, as defined by the Arizona Maternal Mortality Committee, is the death of a woman of any cause while pregnant or within 90 days of termination of the pregnancy, regardless of the duration of the pregnancy at the time of termination or the method by which it was terminated. It would be appreciated if any maternal deaths conforming to this definition would be reported to the Arizona Maternal Mortality Committee secretary at the Arizona Medical Association Building, 810 West Bethany Home Road, Phoenix (telephone 246-8421) or the Committee Chairman, William A. Susong, M.D. (telephone 248-0348).

The Arizona Maternal Mortality Committee met at the Adams Hotel on October 13, 1976 during the meeting of District VIII, ACOG. Of the maternal deaths reviewed, three were due to pneumonia in the third trimester of pregnancy.

The committee summarizes these cases:

CASE 1: A patient in her 20s, Gravida 3, Para 1, Ab 1, was admitted at approximately 37 weeks gestation, in labor, with "flu syndrome" and fever. Chest X-ray on admission was normal. Following 17 hours of labor without satisfactory progress a low transverse Cesarean Section was performed. Cough persisted and increased in severity. Temperature of 103° and rales developed on the third post-operative day, chest X-ray showing evidence of pneumonitis. The course was further complicated by a wound dehiscence requiring secondary closure. Cardiorespiratory arrest occurred in the recovery room necessitating use of a respirator. Tracheostomy was also subsequently required. Thereafter a progressive downhill couse continued, and she expired several days later. Influenza Type A virus was recovered from the sputum. Interstitial pneumonitis consistent with viral pneumonia was found at autopsy.

CASE 2: A Class C diabetic in her mid-20s, Gravida 4, Para 1, Ab 2, was admitted at 32 weeks gestation with back pain. Fever developed shortly after admission, and intravenous Keflin was begun for a suspected pyelonephritis. The following day severe cough, nausea, and vomiting developed. Evidence of pneumonitis was present on chest X-ray. Despite treatment with Chloromycetin, oxygen, and IPPB, the pulmonary condition deteriorated and she had to be intubated with the use of a MA-1 respirator and PEEP (Positive End Expiratory Pressure). Spontaneous onset of labor ensued. A viable premature infant was delivered by low forceps. Respiratory condition deteriorated; tracheostomy was performed 5 days postpartum. Antibiotics were changed when positive cultures for Staphlococci were obtained from the tracheostomy tube. Following a protracted downhill course she expired approximately 1 month after admission. Autopsy was not performed. The primary cause of death was ascribed to viral pneumonitis with secondary bacterial pneumonitis.

CASE 3: A teenage primigravida at 33 weeks gestation was treated for a URI of approximately one week duration. Two days later hospitalization became necessary because of severe dyspnea; T - 101.3°, P - 160, R - 55. Chest X-ray revealed consolidation of the middle and lower lobes of the right lung. Diagnosis was pneu-

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Reprint requests to Dr. Ziehm, 1728 W. Glendale Ave., Phoenix, AZ 85021.

monia or pulmonary embolus. WBC count was 1,000 with 30% polys, 30% bands, and 30% lymphs. On 6 liters of O_2 the pO_2 was 60, pCO_2 was 25-30. Treatment consisted of Ampicillin and nasal oxygen. The following day she was transported to another hospital. On arrival the pulse was 166, respirations 48 and Blood Pressure 84/64. Fetal Heart Tones could not be heard. On admission she was in active labor, the cervix 7 cm dilated. Approximately one hour later a stillborn 3670 gram infant was delivered by low forceps over a midline episiotomy using pudendal block anesthesia. Hypotension and hypoxia persisted following delivery. Her condition rapidly deteriorated and she expired 6 hours following delivery. Autopsy findings were those of severe bilateral stapholococcus aureus bronchopneumonia and severe bronchitis and bronchiolitis consistent with a viral etiology.

The committee was impressed by the fact that, in spite of good and aggressive management, three pregnant women died within a span of a few months due to viral pneumonitis. All three of these patients were young and two of the three had no other significant health problems, one being a diabetic requiring insulin for control. Although viral cultures were positive in only one of these cases, the clinical history and pathologic findings indicated that all three had viral pneumonia as the primary disease leading to death. These cases should make all who care for obstetrical patients more aware of the potential hazard of viral respiratory diseases during pregnancy.

Seminars in Endocrinology and Metabolism

HAIRY WOMEN OR HIRSUTE PATIENT?

MARSHALL B. BLOCK, M.D.

In women, increased quantities of hair, distributed in a normal topographic pattern, is cosmetically bothersome. On the other hand, some conditions associated with the abnormal production of hair, distributed in a male pattern, demands further investigation. How does the physician caring for women patients who complain of hariness appropriately deal with the situation so that abnormal states are detected and appropriately referred, while variations of normal are treated with reassurance? The answer to this question forms the basis of this brief review of hirsutism.

Androgens (hormones producing male sex characteristics) are produced both within the ovary and the adrenal gland in women. Testosterone, the most potent male hormone, in women is produced in extremely small quantities within the ovaries. The levels in females approaches 100 ng/100 ml. which is approximately 1/10 of the level seen in most adult males. Production of testosterone from the ovary appears to be under the control of luteinizing hormone (LH) which is made within the pituitary gland under the control of the hypothalamus. Although the negative feedback regulation of serum LH in women is more complex than in males, very high levels of testosterone will suppress plasma LH values. However, moderate increases in serum testosterone may not produce this effect as they would in men. This is because serum LH in women is under additional combined stimulatory and inhibitory control by serum estrogen levels. Because testosterone is further metabolized within the liver, after it is secreted, into compounds which are not detected by our routine urinary screening measurements, serum determinations are presently the most discriminating way we have to decide whether

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Reprint Requests: 3111 North Fifth Avenue, Phoenix, Arizona 85013 elevated levels of this hormone are present. Unfortunately these measurements are costly and serum levels fluctuate widely throughout the day. These variations create difficulties in assessing whether normal or elevated levels of testosterone are present in women.

The adrenal gland is another major source of androgens. These, however, are made in large quantity but have less biological activity than testosterone. Because they are converted to 17-ketosteroids within the liver after they are secreted, they are more easily measured in the urine. Since the ovaries and adrenal glands are under control of higher centers, i.e., pituitary gonadotrophins (LH, FSH) and corticotrophin (ACTH) respectively, it is not surprising that an attempt to identify the source of excessive androgens in hirsute patients, utilizing this information, has been undertaken. These methods include the use of dexamethasone to suppress plasma ACTH in an attempt to help localize the adrenal gland as the source of the 17-ketosteroid overproduction. On the other hand, a rise in plasma testosterone following stimulation with human chorionic gonadotrophin (HCG) would indicate that the ovary was the source of the androgens.

However, doubt has been cast on this rather simple formulation. The steroid producing cells of the body are pluripotential thus, it is not surprising that cells producing ovarian hormones can be present within the adrenal gland and vice versa. These cells may be under their normal stimulatory and inhibitory influences or they may become so undifferentiated that they can be stimulated or suppressed interchangeably. This is thought responsible for the difficulty in predicting with any degree of accuracy the source of excessive androgens which are being produced in patients who have hirsutism. To adequately predict the source of androgens, catheterizing each gland's venous drainage, to see whether increased concentrations of androgens are present is probably the best procedure now available. However, this is a major undertaking which is costly and not without morbidity so that simpler screening studies are needed to select those patients who should undergo further evaluation.

There are marked variations in the quantity of "normal" hair. For instance, Indian and Black ancestry is linked to less hair than Mediterranean ancestry. The distribution of hair is important. Increased facial hair, especially in the region of the external auditory canals is more pathologic than increased arm hair or hair strands around the areolae. A change from a female to a male escutcheon pattern is likewise a significant finding. Other features of virilism should be noted, e.g., increased muscle mass, clitoromegaly, temporal baldness, changes in voice, etc. In addition, features of cortisol excess which can be associated with increased androgen production from the adrenal gland should be noted.

By far, the most common condition producing mild hirsutism is the polycystic ovary syndrome (formerly known as the Stein-Leveventhal syndrome). Although obesity, oligomenorrhea, infertility and enlarged ovaries were the classic description given to patients with this syndrome, a wider sepctrum of patients with fewer of these symptoms and signs have been included in this category. Classically, laboratory studies may show a mild increase in serum testosterone (less than 200 ng. 100 ml.), a mild increase in serum LH with normal serum FSH and a slightly increased 17-ketosteroid 24 hour urinary excretory pattern (less than 25 mg. 24 hours). In contrast, patients with ovarian tumors such as arrhenoblastomas and Sertoli-Leydig cell tumors usually have more pronounced hirsutism associated with other virilizing features. Serum testosterone levels are usually greatly elevated (greater than 200 ng. 100 ml.) with normal or slightly increased 17-ketosteroid excretion. Serum LH may be high and not suppressed as estrogen deficiency may be present in addition.

Of the adrenal causes of virilization of which hirsutism is one feature, malignant tumors characteristically present first with these features while benign lesions like adrenal hyperplasia or an adrenal adenoma usually give "cortisol excess" clinical findings first and only later produce symptoms of virilization. In the latter conditions, 17-ketosteroid excretion is markedly increased, and in addition, 17-Hydroxy steroid excretion is also elevated. Serum testosterone levels can be normal or slightly increased in these conditions.

Thus, at the present time, basal 24 hour urinary 17-ketosteroid and 17-Hydroxy steroid excretion and plasma testosterone measurements, performed in reliable laboratories, are needed to help decide which patients should be referred for more sophisticated studies. The therapeutic benefits of this approach can be extremely rewarding both for the physician and patient.

Seminars in
Chest Medicine

ASTHMA THERAPY — 1977

BERNARD E. LEVINE, M.D.

This continuing series of articles entitled "Seminars in Chest medicine" will attempt to keep the reader abreast of developments in the broad field of pulmonary diseases. The format used will be that of brief succinct reviews written by the editors as well as guest contributors. Areas of controversy as well as practical chest medicine will be explored. We hope that these reviews will be of value in promoting continuing education for certification examinations as well as a forum for new and controversial issues. The editors welcome comments and discussion from our readers.

ROBERT J. CLARK, M.D.
LYNN M. TAUSSIG, M.D.
WILLIAM C. WEESE, M.D.

Important new therapeutic agents are becoming available for the treatment of bronchial asthma, which will greatly change our approach to the treatment of wheezing disorders. This paper will review earlier approaches to asthma, discuss those of major importance and describe the newer drugs and their role in asthma therapy. For this discussion, it is convenient to classify wheezing syndromes into (a) sporadic asthma, (b) frequent asthma, and (c) continuous asthma. Sporadic asthma is defined as wheezing occurring no more frequently than twice a week and quickly reversible with therapy. In this type of asthma, nearly any of the available bronchodilators (oral or inhaled) will be effective. Frequent asthma refers to daily wheezing; for it, the patient should have a regular medication schedule. Frequent asthma may be seasonal or perennial. As asthma requires increasing medication and becomes unremitting, it is termed continuous asthma. Continuous asthma not responding to appropriate home therapy can lead to status asthmaticus and requires office or hospital therapy.

In addition to pharmacologic agents for asthma, there are several general principles of treatment. First is identification and avoidance of irritants and allergens whenever possible. Appropriate hydration will minimize mucus plugging and expectorants may be of value in patients with thick, tenacious sputum. Immunotherapy may also play a role in wheezing disorders but its role in the adult asthmatic is yet to be established.

In the past, frequent asthma has been treated with both oral and inhaled medications. The former include both theophylline preparations and combination drugs containing theophylline, ephedrine, and a sedative or tranquilizer (e.g. Marax, Tedral). These combined products are not optimal medications, especially for adults. The amount of theophylline in these preparations will not create an effective blood level. The sedative in these drugs is usually not strong enough to overcome the excitatory effects of ephedrine, and finally ephedrine is no longer the best oral adrenergic agent available. Inhaled therapy has consisted either of isoproterenol (Isuprel), racemic epinephrine (Vaponefrin), or isoetharine (Bronkosol) from hand-bulb nebulizer, freon-powered nebulizer, or compressor-powered nebulizer. Now, newer inhaled bronchodilator drugs have significant advantages in terms of duration of action and selective bronchodilator versus cardiac (beta-2 versus beta-1) effect.

Theophylline remains an effective agent. Development of and clinical availability of rapid theophylline assays have increased our understanding of the clinical usefulness of this drug. An optimal theophylline blood level is 10-20 micrograms per milliliter of blood (mcg/ml). In this range, most patients with reversible airways obstruction achieve bronchodilation. Blood levels above 20 mcg/ml are often associated with symptoms of toxicity (nausea, headaches, etc.). Blood levels below 10 mcg/ml are significantly less effective in bronchodilation. In order to achieve an effective theophylline blood level in the adult, oral anhydrous theophylline doses of approximately 1000 to 1400 mg per day are required (1200-1600 mg per day of aminophylline). Individual patient needs vary because of differential rates of theophylline absorption and metabolism. When a question exists whether a patient is getting too little or too much theophyl-

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For Reprint requests, Dr. Clark, 2910 N. Third St., Phoenix, AZ 85012.

line, blood levels should be obtained; these are available in the Phoenix area. While aminophylline tablets are the least expensive asthma therapy, other theophylline derivatives may be better suited for an individual patient because of gastrointestinal tolerance. Long acting theophylline preparations have the specific advantage of maintaining more stable theophylline blood levels. Thus, theophylline is a safe and effective medication for the older pediatric and adult wheezing population, and will continue to serve as an important component of long-term programs for patients with frequent or continuous wheezing.

Newer adrenergic agents have been developed for oral and inhaled bronchodilator use. These drugs were created by altering the side chain in the basic adrenergic molecule so as to diminish relative alpha and beta-I effects and increase the duration of action. These medications act longer and produce fewer cardiac and systemic side effects than ephedrine and isoproteronol. Terbutaline (Brethine, Bricanyl) is a good example of this type of agent. It is effective orally (now available), by inhalation (not yet released), and parenterally. Some studies have reported up to six hour duration of bronchodilator action after inhalation of aerosolized terbutaline. Oral terbutaline tends to cause significant peripheral tremor. Beta-II stimulants tend to decrease twitch tension in skeletal muscle thus leading to tremor. In certain patients, therefore, the usefulness of this drug may be limited. If the drug is effective, but the twitching intolerable, addition of vistaril or Valium may be helpful.

In establishing a treatment regimen for frequent asthma, oral theophylline, a beta-II adrenergic agent, or both may be needed for wheezing control. An obvious general rule in selecting a program is that if either drug fully reverses the bronchospasm, the best tolerated drug is chosen. In one study, aminophylline alone produced bonchodialtion; terbutaline alone caused slightly more, but the addition of aminophylline to terbutaline did not improve upon the effects of the latter. Within this particular group of patients, terbutaline alone would have been adequate bronchodilator therapy. In some patients, however, it may be necessary to use more than one oral drug and one or more inhaled drugs. Clinical and pulmonary function testing may be important to plan the most effective, best tolerated drug regimen for individual patients.

Most beta adrenergic stimulants (isoetharine, metaproterenol, salbutamol, terbutaline) either are or will soon be available in freon-powered nebulizers. The use of an inhaled bronchodilator by this route is a somewhat controversial subject. These nebulizers are valuable tools and may be extremely important for many patients with wheezing, but their abuse can be dangerous. Over-frequent use may cause local irritation and worsen patients with wheezing syndromes. The long duration of action of these agents increases the danger of drug over-dosage with nebulizer abuse. Therefore, freon-powered nebulizers should be used only in those patients with whom rapport and communication are adequate so the patient understands how he must and must not use the nebulizer. When used appropriately, freon-powered nebulizers are important therapeutic devices for reversible bronchospasm.

In many patients, especially those with mucus plugging, hydration and bronchodilation are important, and use of inhaled bronchodilator agents in a dilute solution administered by a compressor powered nebulizer is appropriate. Multiple breaths of dilute aerosol from such a device (e.g. Maximyst, Pulmonaid) may distribute bronchodilator agents better than a single breath from a freon-powered unit. This may be important in patients with severe obstruction and decreased inspiratory capacity. Unfortunately, newer medications (terbutaline, salbutamol) are not yet available in a form easily delivered by a compressor powered nebulizer.

Disodium cromoglycate (Aarane and Intal) has been available for four years and is dramatically effective for many asthmatics, especially patients with exercise-induced or pollen sensitive asthma. This drug attaches to the mast cell, stabilizing it so it does not disintegrate and release histamine and other bronchoconstrictor substances. In some patients, this drug alone may suffice; it should be tried before long-term steroids are utilized. It is important to remember that this is an asthma preventative, and is not effective for stopping asthma attacks once they have begun. Because it is a dry powder and may be an irritant, it should not be used in the presence of acute respiratory infections or status asthmaticus.

Corticosteroids should be the last drugs to be added to an asthmatic program, and the first drugs to be removed. An approach to steroid therapy in asthma is summarized below. First, in status asthmaticus, large doses of steroids should be used on a round-the-clock basis to maintain optimal steroid blood levels. After treatment of an acute exacerbation, steroids should be tapered and stopped, ideally within a 10-12 day period. If long-term steroids are required, they should be used at the lowest dose and least frequent dosage interval possible. Steroids should be given preferably on an alternate day basis in the morning, and, if this is not adequate, as a single morning daily dose. A pattern of morning dosage will suppress adrenal function less than a single dose of steroids at night or split daily dosage.

Aerosols have revolutionized the use of corticosteroids. Beclomethasone dipropionate (Vanceril) has been used for six years in Great Britain. This drug, inhaled from a freon-powered nebulizer, is topically active in the lungs and has minimal adrenal suppression in the dosage usually required. The drug has an excellent record of use; except for infrequent oral candidiasis and hoarseness, no significant side effects have been ascribed to it. In patients who have been treated with oral steroids, normal adrenal function returns within weeks to months. During the period of substituting inhaled for oral steroids, however, one must observe the patient for signs and symptoms of adrenal insufficiency. An excellent protocol for tapering from oral corticosteroids was recently published.[1]

During the past 18 months, we treated 37 asthmatic patients with triamcinolone acetonide, a drug similar to beclomethasone. Results with this inhaled steroid have been dramatic. Patients have been able to discontinue oral steroids after periods as long as 20 years on therapy. For long-term therapy of the steroid-dependent asthmatic, inhaled steroids are clearly a major advance in our pharmacologic approach.

Much current research concerns the nature of the asthmatic response and its pharmacologic control. Inhaled atropine and atropine-like drugs appear to be effective in control of wheezing and revive an old class of anti-asthmatic medications. Bronchodilator effects of prostaglandins are being studied. Research on the wheezing-response itself will help us better utilize the new drugs to treat more specially defined wheezing states.

SUMMARY

The approach to the asthmatic continues to include avoidance of irritants and allergens, prompt treatment of respiratory infections, adequate hydration, adequate physical activity, and physical and emotional rest. Therapy may mix oral and parenteral theophylline along with newer beta-II adrenergic drugs and anticholinergic agents in dosages to provide maximum bronchodilation with minimal side effects. Inhaled bronchodilators will continue to represent a valid mode of therapy for wheezing disorders. The use of freon-powered nebulizers should be limited to patients who are trustworthy and will not abuse them. Cromolyn sodium is an effective, preventative agent with minimal side effects in a significant percentage of patients. Inhaled steroids will hopefully replace and certainly reduce oral steroid needs in a great majority of patients. Finally, increased understanding of the pathophysiology of asthma will lead to continuing improvement in our approach to this often extremely complex clinical problem.

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Grand Rounds

PEMPHIGUS — A REVIEW

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Pemphigus is a rare chronic blistering disease of the skin and mucous membranes which has been given inadequate attention in general medical literature. Thus, for example,

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the mortality rate and incidence are quite similar for the two diseases, malignant melanoma and pemphigus, yet the former receives many times the medical attention. With this in mind we would like to present our recent experience with pemphigus. During the last three years we have seen eight new patients with this diagnosis. Five of these eight patients have had most of their management at the University of Arizona Health Sciences Center and will form the basis for this Grand Rounds presentation.

REPORT OF CASES

Case 1

A 33-year-old Mexican American female was first seen in June 1975 because of a one year history of oral and pharyngeal erosions. Bullous lesions had recently appeared on the shoulders, chest, abdomen, back and extremities. She had experienced difficulty in swallowing and had had significant weight loss. On examination, erosions were noted in the mouth and pharynx. Bullous lesions in various stages of healing were scattered on the chest, back, neck, trunk and extremities. A skin biopsy was consistent with pemphigus vulgaris. Immunofluorescent staining was positive for pemphigus antibodies. Skin tests were positive for mumps and tuberculosis but negative for coccidioidomycosis. She was treated with 150 mg of prednisone every day for three weeks. Over the following months, her prednisone dose has gradually been reduced to 100 mg every other day. At the present time she is healthy and her pemphigus is under complete control.

Case 2

A 30-year-old white male was seen in January 1975 because of a six month history of recurrent, painful oral blisters. Two weeks prior to examination, blisters were first noted on the chest and axillae. On examination of the mouth there were multiple eroded areas covered with pseudomembranes. Several small bullae were present in the axillae. A biopsy of an axillary lesion showed pemphigus vulgaris. Direct and indirect immunofluorescent studies were positive for pemphigus. He was initially treated with 100 mg of prednisone every day and the dose was gradually decreased to 60 mg every day. At that point, the dose was converted to 120 mg every other day. His oral and skin lesions cleared but a granuloma became apparent on X-ray of the chest. Before this could be further evaluated, he moved to Boston. A recent letter from his physician stated that he developed disseminated coccidioidomycosis and died.

Case 3

A 34-year-old Mexican American female was first seen in November 1975 with a three year history of bullous eruptions of the face, shoulders and trunk. A diagnosis of pemphigus had been made and she had been treated with varying amounts of prednisone. Her course had been marked by poor control and by recurrent bacterial and herpetic infections. Examination revealed crusted weeping lesions in the scalp with rare small vesicles on the trunk. No oral lesions were present. A skin biopsy was consistent with pemphigus foliaceus. Immunofluorescent studies were positive for pemphigus antibodies. Her prednisone dose was increased to 200 mg per day at which point adequate control was achieved. Dicloxacillin has been used on several occasions to control cellulitis of the scalp. At the present time she continues to have some scalp involvement on 120 mg of prednisone every other day.

Case 4

A 53-year-old Mexican American female was first seen in July 1975. She had a seven month history of vesicular lesions on the legs and a three month history of larger, bullous lesions on the face, chest, back and buttocks. On examination, bullous lesions involved the entire body with predilection for the medial surface of both arms, the intertriginous areas, and face. No oral lesions were present. A biopsy from the forearm showed the changes of pemphigus foliaceus. Immunofluorescent studies, done on one occasion only, were negative. She was initially treated with 250 mg of prednisone every other day. Over the subsequent two months, worsening of her disease necessitated the use of methotrexate 25 mg intramuscularly every two weeks. Control was still not achieved and intramuscularly administered gold was substituted for the methotrexate. At this point she is healthy and is free of pemphigus lesions on 70 mg of prednisone every other day and gold sodium thiomalate 50 mg weekly.

Case 5

A 65-year-old white male was first seen in July 1976. He had a seven month history of oral erosions and a three month history of bullae on the neck, back and upper trunk. He had lost 10 pounds since his illness began. On examination, multiple erosions were found in the mouth and on the upper trunk. A few small intact vesicles were present on the trunk. A biopsy from the chest showed pemphigus foliaceus. Immunofluorescent studies were positive for pemphigus antibody. He was initially started on 120 mg of prednisone every day. This was increased to 240 mg daily after three weeks of therapy with minimal improvement. At about this time he developed a cough and fever and a chest X-ray showed diffuse granulomatous disease. A culture from an open lung biopsy grew *Coccidioides immitus*. Steroids were quickly tapered to maintenance levels and he was started on intravenous Amphotericin B and intramuscular gold injections. At the present time his health remains seriously compromised and his pemphigus is only partially controlled.

EPIDIEMIOLOGY

Age. Pemphigus is generally a disease of middle and late adult life; the median age at onset occurs in the sixth decade. Nevertheless, the range in age is wide varying from 12 to 88.[1-3] Except for the Brazilian variant, pemphigus in childhood is relatively uncommon.

Sex. Various studies have reported a slight excess of men over women but the sex distribution in the three recent large series showed 118 men and 111 women.[1-3]

Incidence. The prevalence of pemphigus is not known with certainty. In one older study, patients with pemphigus accounted for slightly less than 1% of all visits to a dermatology clinic at a major referral hospital.[4] A very rough annual incidence rate of 0.135 cases per 100,000 population was determined for Jerusalem—an area of known high prevalence.[6] Our eight new cases in three years, all but one of whom was less than one year in duration, suggest an annual incidence rate of approximately 0.5 per 100,000 population here in southern Arizona. These eight cases have accounted for approximately 0.2% of all visits to the University Dermatology Outpatient Clinic. These figures are roughly comparable to our experience with malignant melanoma.

Race, Nationality, and Religion. People of all races and all geographic locations are known to develop pemphigus but some groups seem to have a higher than expected predilection for the disease. Jews seem to be particularly susceptible.[1-3 6] In one large series Jews accounted for 74% of the pemphigus vulgaris patients even though they made up only 8% of the hospitalized patients at that institution.[7] Even among Jews there is variability; one study suggests that Ashkenazi Jews apparently are four times more susceptible than are Sephardic Jews.[6] Brazilians also have a very high predilection for pemphigus but, in this instance the susceptibility seems better related to malnutrition and low socioeconomic class rather than to a common national background. Three of our eight patients were Mexican Americans, a proportion double that of our overall clinic population. However, in a large study from the Los Angeles area Mexican Americans did not seem to be unduly represented[3]

CLINICAL FEATURES

Historically, four types of pemphigus have been recognized but critical review suggests that there are only two basic groups: 1) patients with supra basalar bullae (pemphigus vulgaris) and, 2) patients with mid epidermal bullae (pemphigus foliaceus). These two groups are easily separated by conventional skin biopsy and there appears to be little if any cross-over from one group to the other.

Pemphigus Vulgaris. Pemphigus vulgaris is the most common type of pemphigus and it accounts for approximately 80% of all patients with pemphigus in three recent, large series.[1-3] Approximately 60% of patients with pemphigus vulgaris develop their initial lesions in the mouth;[1-3] oral lesions are eventually found in 80 to 90% of these patients.[1 3 6] Other mucous membranes such as the vulva, conjunctiva, pharynx, and larynx also may become involved.[1] Lesions in the two latter locations account for much of the debilitation seen in this disease. The lesions on the mucous membranes are rarely seen as intact blisters because of the fragility of the blister roofs. The examining physician will instead see areas of irregularly shaped oral erosions which bleed easily. These erosions show little tendency to heal even when followed over a period of weeks. The large size of the erosions, the involvement of the posterior mouth, and the slow healing help to differentiate the oral lesions of pemphigus vulgaris from other ulcerating diseases involving the mouth.

Non-mucosal, cutaneous lesions consist of fragile, flaccid bullae which generally occur within a short time (average five months) after the appearance of the oral lesions.[3] These blisters arise from noninflamed skin and vary in size from 1 to 10 cm. Because of their fragility the bullae also break easily, resulting in large areas of superficial erosion. The weeping and crusting found over the erosions may mimic the appearance of widespread impetigo. Slow healing of the eroded skin results in the eventual involvement of huge areas of the body. Any part of the body may be involved in pemphigus vulgaris but there is some predilection for the upper trunk and for pressure points, groin and axillae. When healing does occur (whether spontaneous or as a result of treatment), no scarring is present but there is a varying degree of post-inflamatory hyperpigmentation. The Nikolsky sign, defined as easy separation of epidermis from dermis on lateral pressure with the finger leaving a new erosion, is positive in pemphigus.

Approximately 2 to 5% of patients with pemphigus vulgaris eventually develop pemphigus vegetans which has the appearance of verrucous or vegetative nodules in the eroded areas.[1-3] Small pustules are often seen around the vegetations. Pemphigus vegetans is probably decreasing in frequency now that definitive treatment for pemphigus vulgaris is carried out early in the course of the disease.

Pemphigus Foliaceus. Pemphigus foliaceus acounts for approximately 20% of all patients with pemphigus.[1-3] In this disease, oral lesions are rarely seen and if present, are considerably less troublesome than those found in pemphigus vulgaris. The initial cutaneous lesions occur on the chest and shoulders but the face and scalp may also be involved early. Later, widespread areas of the trunk and extremities may become involved. Intact blisters are seldom clinically apparent as the blister roofs are even more fragile than those found in pemphigus vulgaris. Instead, erosions covered with relatively dry crust or yellow scale are the predominant feature. These lesions do not bleed as easily when traumatized as do those found in pemphigus vulgaris, probably owing to their more superficial location in the epidermis.

Several subtypes of pemphigus foliaceus exist. In pemphigus erythematosus, the lesions are confined to sun exposed areas and look strikingly like the lesions of systemic lupus erythematosus. Moreover, sun exposure tends to worsen the disease. In fogo selvagem, the Brazilian type of pemphigus foliaceus, there is relatively little morbidity and the disease tends to occur epidemically among younger family members. In the third subtype of pemphigus foliaceus, individual vesicles are small and grouped, greatly resembling dermatitis herpetiformis.[8] Microscopically eosinophilic abscesses without acantholysis occur within the epidermis. However, these patients, over a period of time, go on to develop more typical features of pemphigus foliaceus.

LABORATORY FINDINGS

Histopathology. The histopathology of both types of pemphigus is characteristic, if not pathognomonic. In pemphigus vulgaris, the intraepidermal bullae are located low in the epidermis just above a single layer of basal cells. The basal cells of the epidermis remain attached to the dermis but cells in the next serveral layers separate from one another due to the loss of intercellular bridges and appear as loose, rounded up cells within the bullae (acantholysis). Irregular upward growth of the dermal papillae lined by a single layer of basal cells is also frequently seen. A few inflammatory cells may be present within the bullae and these are mainly lymphocytes and eosinophils. The dermis shows a moderate inflammatory infiltrate, again mostly lymphocytes and eosinophils. Electron microscopy shows damage to the tonofilament-desmosome complex with detachment of the tonofilaments and disintegration of the desmosomes. Immunofluorescent studies show the presence of circulating auto-antibodies to the intercellular substance of stratified epithelium. The antibody titer is proportional to the severity of the disease. This IgG antibody, together in some cases with complement, is found deposited between the epidermal cells in biopsy specimens taken from the edges of blistering lesions.

Pemphigus foliaceus in contrast to pemphigus vulgaris shows an intraepidermal cleft (or more rarely a bullae) located in a much more superficial location. In fact, the area of separation often lies just beneath the stratum corneum. Acantholysis is present as well as dyskeratotic changes of the granular layer. The dermis shows a moderate number of inflammatory cells, including eosinophils and lymphocytes. Electron microscopy shows abnormalities similar to but less severe than those seen in pemphigus vulgaris. Immunofluorescent studies are similar to those found in pemphigus vulgaris but in some instances the antibody to the intercellular substance is found only in the subcorneal layer. Further, in those patients with the subtype of pemphigus foliaceus known as pemphigus erythematosus, LE type antibody may be found deposited at the dermal-epidermal junction. Some of these latter patients also have circulating antinuclear antibody.

Other Laboratory Examinations. No ohter characteristic laboratory findings occur. Eosinophilia may be present but is not distinctive. Hypoalbuminemia and a nonspecific increase in alpha 2 globulins may occur in chronic cases with inadequate treatment. Serum IgE levels are normal[9] as are serum complement levels.[10] Blister fluid levels of complement, however, may be reduced.[11]

PATHOGENESIS

Pemphigus is believed to be an autoimmune disease for the following reasons: 1) IgG antibody to the intercellular areas of the epidermis is found both circulating within the serum and deposited within the epidermis of skin lesions.[12] 2) This antibody is deposited specifically at the site where the histogic damage in pemphigus is seen.[12] 3) Antibody of this type is seen only rarely in other diseases.[13] 4) The titer of circulating antibody corresponds to the severity of the disease and it decreases with successful treatment. 5) Complement levels are decreased in the blister fluid. 6) Under certain circumstances, injection of this antibody into laboratory animals may result in a blister similar to that seen in human patients.[14] 7) Pemphigus is found more often than expected in association with other autoimmune diseases (see below). 8) Pemphigus responds to steroids and nonsteroidal immunosuppressive therapy.

Unfortunately, attempts to identify the antigen which leads to production of the antibody discussed above have not yet been successful. Currently it is believed that this antigen may be a glycoprotein component of the outer leaflet of the epidermal cell wall.[15-17] This antigenic substance may also exist between and within the epithelial cells of the thymus—suggesting an immunologic problem which might involve T cells as well as B cells.[18]

Genetic as well as autoimmune factors also play a role in the development of pemphigus and are reflected in the predilection for certain groups to have higher than expected prevalence of the disease (see above). Moreover, there are at least two reports suggesting a possible association between pemphigus and certain histocompatibility antigens.[19,20]

Finally, environmental factors are also important. Lesions tend to arise in areas of skin that are easily and frequently traumatized. And, at least in pemphigus foliaceus, new lesions can be induced experimentally by exposure of the skin to ultraviolet light.[21]

ASSOCIATED DISEASES

Certain autoimmune diseases have been reported to occur with unexpected frequency in patients with pemphigus. Myasthenia gravis has been noted to co-exist with pemphigus in at least 12 patients.[22,23] In all instances, the myasthenia gravis preceded the appearance of the pemphigus. Of further interest is the fact that pemphigus-type, antiepithelial antibody was demonstrated in 15% of one series of patients with myasthenia gravis who otherwise had no clinical evidence of pemphigus.[24]

Lupus erythematosus has likewise been linked with pemphigus. In one study of 24 pemphigus patients, antinuclear factor was present in the sera of seven.[25] Moreover, in another study, LE type antibody was demonstrated at the dermal-epidermal junction of cutaneous lesions in five of six patients with pemphigus erythematosus.[26] One of these five patients had antinuclear factor and a positive LE cell preparation.

Although both lupus erythematosus and myasthenia gravis can occur in patients with pemphigus vulgaris, both of these diseases are seen far more frequently in patients with pemphigus foliaceus—particularly those with the subtype of pemphigus erythematosus. No explanation for this phenomenon exists.

Tumors may also occur with greater than expected frequency in patients with pemphigus.[5,27] Thus, benign and malignant tumors were found in 12% of one series[5] and in 4% of another.[1] More than half of these tumors were related to the hematopoietic and reticuloendothelial systems. Seven of the 26 tumors were thymomas[5] and two more thymomas have subsequently been reported.[23,28]

PROGNOSIS

Good data regarding the natural history of pemphigus are available from studies published prior to the era of corticosteroid treatment.[29] The mortality rate in these "untreated" patients was essentially 100% in patients with pemphigus vulgaris and only a little lower in those with pemphigus foliaceus. Patients with pemphigus vulgaris died within an average of 18 months whereas those with pemphigus foliaceus had a more protracted course lasting five years or longer.

Treatment with steroids and other immunosuppressive agents has led to a gradual improvement in the prognosis. This improvement has allowed recognition of at least three other factors which influence prognosis. The first is age.[1,2] The mortality rate is considerably higher for those individuals who develop the disease after age 50. The second is duration.[1] More than half of the patients who will die with pemphigus do so within the first year; thereafter, the survival curves level and five year survival is practically tantamount to cure. The third is severity of the disease.[1,2] Patients requiring the largest doses of steroids continue to have a mortality rate of up to 75 percent. Unfortunately, it is not clear what proportion of this high mortality rate is due solely to the severity of the disease and what proportion is due to the complications of vigorous therapy.

THERAPY

Corticosteroid Therapy. Four series of reasonable size have been published in the last five years reviewing the results of treatment with corticosteroids.[1,3-30] The therapeutic program in all four studies was similar and consisted of the currently preferred high initial dose approach originally recommended by Lever. In these studies there were 183 patients with pemphigus vulgaris. Sixty-five of these patients (35%) died of causes related to the disease or its treatment. Five year survival figures could not be determined accurately for all four studies but it was clear that mortality decreased remarkably for those patients who survived beyond the first year. Most of the patients who died were older than 50 at the time their disease became apparent and there was a considerable excess of deaths in those patients requiring more than 100 mg of prednisone per day to control the disease. The outlook for pemphigus foliaceus was better in that only 13 or 53 patients (25%) died form the disease or complications of treatment.

Patients who survived five years generally required small amounts of steroids (20 mg of prednisone or thereabouts) for maintenance and 15% of the patients could be considered cured as they had undergone long remissions without therapy.

The steroid complication rate was very high with up to 85% of all of the treated patients having one or more complications. Infection was the most frequent serious complication. Bacterial and fungal infections have been a problem in our patients and it should be specifically noted that two of our patients have developed coccidioidomycosis.

As might be expected, the pemphigus antibody titer decreased to very low levels or became totally inapparent in those patients who were in remission. Increased levels of antibodies frequently preceded exacerbations.

There has been one small study where alternate day steroid therapy was attempted.[31] Seven patients (maintained under good control with prednisone in doses of as high as 60 mg a day) were successfully converted from daily to every other day dosage. Three additional patients mild disease achieved remissions when they were treated initially with prednisone 160 mg every other day. In these three latter patients, tests of cell mediated immune response and pituitary adrenal function were normal even though they were taking 90 to 150 mg of prednisone every other day at the time the tests were performed.

Methotrexate. There have been two studies where patients with newly diagnosed pemphigus were initially treated with methotrexate.[30,32] The dosage used was 25 to 50 mg per week. Those patients with pemphigus foliaceus and pemphigus erythematosus improved considerably but in patients with pemphigus vulgaris oral ulcerations persisted.

Three studies discuss the use of methotrexate for those patients who are being maintained on steroids.[30,32,33] Two groups found that they could both control the disease and reduce the maintenance steroids whereas the third group was less successful primarily because of the development of side effects secondary to the methotrexate. Persistent, oral ulcerations were noted in all three studies.

In summary, Lever,[30] the individual who probably has the most experience with the use of methotrexate, states that methotrexate is not appropriate for *initial* therapy in patients with severe pemphigus. However, he does believe that methotrexate can and should be used to reduce steroid side effects once the disease is controlled and maintenance levels of steroids have been reached. In support of this he quotes one of the lowest mortality figures (8%) for patients treated under any program.

Azathioprine. There has been only one major study regarding the use of azathioprine in pemphigus.[29] In this study five patients with newly diagnosed pemphigus were treated with azathioprine in doses of 150 to 250 mg per day. Three of these five patients plus an additional 11 patients found by the authors in a review of the literature were satisfactorily treated.

These same authors[29] describe 15 patients (two of their own and 13 from other studies) in whom azothioprine was successfully used to lower the maintenance dose of steroids for patients in steroid-induced remissions.

No serious side effects except reversible leukopenia were seen in any of the azathioprine treated patients. No one currently advocates the use of azathioprine for the induction of remissions but Roenigk and his group believe that azathioprine can and should be used to reduce steroid side effects in patients on maintenance therapy.[29] They believe that azathioprine is preferable to methotrexate as it is reported to have less of a detrimental effect on the healing of experimentally induced wounds.

Cyclophosphamide. Based on a single report, cyclophosphamide given intravenously cannot be used to induce remissions.[34] However, in 13 patients with steroid-induced remissions, steroids could be reduced or even discontinued when cyclophosphamide was used in dosages of 50 to 200 mg per day.[34 35] Side effects were not severe and consisted mainly of reversible leukopenia and alopecia.

Gold. There has been one large series of patients treated with intramuscularly administered gold.[36] Fourteen of 18 patients were successfully treated using either gold alone (mild disease) or gold plus prednisone (severe disease). Eight of the 14 patients are in prolonged remissions with no therapy of any kind. The gold was given as gold sodium thiomalate, 50 mg weekly. The average total dose for those in remission was 1000 mg and average total dose for those still under maintenance therapy has been 1762 mg. Toxicity required the discontinuation of gold in two patients.

The method by which gold therapy is effective is unknown. It does not significantly decrease the synthesis of pemphigus antibody nor does it appear to interefere with the interaction of pemphigus antibody and its antigen.[36] It is known that gold decreases prostaglandin synthesis and also decreases the activity of lysosomal enzymes containing sulfhydryl groups but just how this would effect pemphigus is not entirely clear.[37]

THERAPEUTIC RECOMMENDATIONS

For initial therapy of patients with moderate to severe disease, high dose prednisone therapy is the treatment of choice since all other agents require weeks or months of treatment before peak efficacy is reached. Gold therapy can be started concomitantly with the steroids as the side effects do not appear to be additive whereas this appears not to be true for methotrexate, azathioprine or cyclophosphamide.

In patients with mild initial disease either methotrexate, azathioprine, cyclophosphamide or gold can be successfully used but improvement may not be seen for many weeks. These four agents can also be used for patients in steroid maintained remissions. This approach (or the conversion to alternate day steroid therapy) will greatly reduce steroid side effects which are now the greatest cause of death in patients with pemphigus. Adequate treatment such as described above should result in a mortality rate of 10% or less—a vast improvement over the situation which existed just a few short years ago.

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Drug Therapy Problems

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Most practitioners rely upon their own reading plus other external sources to assist them in their quest to remain abreast of the biomedical literature. This feature is intended to provide finite information, to answer some questions, and to stimulate awareness of the availability of an unbiased source of biomedical information. The format will include: questions and answers, with the questions being provided by the readers and/or users of our service; abstracts from the literature; brief descriptions of newly-marketed items; brief discussions of new innovations in therapy; and short exercises regarding specific drug products.

Abstract of Interest: Friedman, W. F., *et al:* Pharmacologic Closure of Patent Ductus Arteriosus in the Premature Infant, *N Engl J Med* 295: 526-529, 1976.

"Six consecutive premature infants received a single oral (2.5 mg/kg) or rectal (5.0 mg/kg) dose of indomethacin, a potent prostaglandin synthetase inhibitor. Within 12 to 24 hours permanent disappearance of all of the clinical symptoms and physical, radiographic and cardiac ultrasound signs attributable to substantial left-to-right shunting though a patent ductus arteriosus had occurred, presumably by constricting the vessel. A transient reduction in renal function was observed in two infants in whom sustained ill-effects did not occur."

Abstract of Interest: Heymann, M. A., Rudolph, A. M. and Silverman, N. H.: Closure of the Ductus Arteriosus in Premature Infants by Inhibition of Prostaglandin Synthesis, *N Engl J Med* 295: 530-533, 1976.

"Eighteen premature infants with patent ductus arteriosus were treated with aspirin (three infants) or indomethacin (15 infants) to block prostaglandin synthesis and pharmacologically close the ductus arteriosus. After aspirin (20 mg/kg every six hours for four doses) the ductus closed permanently in one infant within 24 hours; in another, constriction occurred with clinical improvement and the third infant did not respond. No complications were encountered with aspirin. In five infants given 0.3 mg/kg of indomethacin, complete closure occurred within one day: two of them who received three doses had an elevated serum creatinine for one week. Ten infants received 0.1 mg/kg of indomethacin and closure occurred within 24 to 30 hours in eight. One had a soft murmur for four days and one did not respond to two doses of indomethacin. Renal function was unaltered in infants receiving less than 0.3 mg/kg."

Abstract of Interest: Mani, V., *et al:* Treatment of Ulcerative Colitis with Oral Disodium Cromoglycate, *Lancet* i, 439-441, 1976.

"Twelve patients (aged 17-69 years) with ulcerative colitis of six to ten years duration, whose disease process was judged to be in remission or with very slight symptoms were studied. Previous drug therapy was continued including salicylazosulfapyridine, but no patient was taking systemic steriods or azathioprine. Patients were treated for six months each with oral disodium cromoglycate (2 gm/day) or placebo in a double-blind cross-over trial. Patients experienced a significant improvement in their sense of well-being and in sigmoidoscopic and histologic appearances during active drug treatment. Two patients developed hepatic abnormalities, although a direct cause and effect relationship was not established. Authors' interpretation of results is guarded with call for further studies."

Abstract of Interest: Novak, E.: Unfavorable Effect of Atropine-Diphenoxylate (Lomotil) Therapy in Lincomycin-caused Diarrhea, *JAMA* 235: 1451-1454, 1976.

"In this double-blind, randomized study, 200

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normal subjects received a three-day course of one of five treatment regimens: lincomycin hydrochloride monohydrate injection (sterile solution, 300 mg/ml) with two tablets of either placebo, a mixture of atropine sulfate and diphenoxylate hydrochloride (Lomotil), an aspirin-phenacetin-caffeine (APC) combination or the latter with codeine, or an injection of saline with two placebo tablets Gastrointestinal irritation was most prominent in subjects receiving lincomycin with atropine-diphenoxylate and lincomycin with APC plus codeine ($P < .05$). Decreased intestinal motility from atropine-diphenoxylate or codeine may increase the contact time between the lincomycin (or its metabolites) or some developing toxic substances and the mucosal epithelium. The use of atropine-diphenoxylate or codeine in treating lincomycin-induced diarrhea may be questionable (Author's abstract).

Abstract of Interest: Williams, R. L., *et al:* Influence of Acute Viral Hepatitis on Disposition of Pharmacologic Effect of Warfarin, *Clin Pharmacol Ther* 20: 90-97, 1976.

"Five subjects (aged 24-33 years) who had no evidence of acute viral hepatitis were selected for the study. Subjects had no history of alcohol or drug abuse. Subjects received a single dose of warfarin (15 mg in 20 ml water) during and after recovery. Mean (± SD) clearance, volume of distribution and half-life of the drug were 6.1 ± 0.9 ml/hr/kg, 0.09 ± 0.04 L/kg and 23 ± 5 hour., respectively during the acute period. After apparent recovery, observed values were 6.2 ± 0.7 ml/hr/kg, 0.21 ± 0.02 L/kg and 25 ± 3 hr. These differences were not significant. Pattern of renal elimination of warfarin metabolites and drug protein binding did not change between the two phases. During the acute period of illness, prothrombin time increased in two of the five subjects, but remained within normal limits in all participants during their recovery period. The authors conclude that warfarin disposition may not change as a consequence of mild or moderate hepatic impairment."

The A.M.S.A. Report

AMSA ACTIVITIES

On behalf of the Arizona Chapter of the American Medical Student Association (AMSA) I would like to thank ArMA for its continuing support and recognition of AMSA. I would also like to outline the activities we have planned for this year.

At the first AMSA meeting of the scholastic year it was announced that ArMA would welcome student representation on its committees. This was met with much enthusiasm. In fact there were so many more people interested in serving as delegates than there were positions, that we formed subcommittees to back of the elected representatives.

Just recently the selection of student delegates to the AMA convention in Philadelphia was completed. I will serve as delegate and Rob Wilson will serve as alternate. Thank you for giving us these opportunities to participate in both the state and national medical associations.

The first activity that our chapter has undertaken this year is that of health career counseling in the local high schools for senior students. Since "pre-med" is a major that many students consider, it was felt by our membership (from our own experiences) that there was a need for access to accurate information about pre-medical and medical education. Tucson School District One has approved our offer and we hope to institute the service in the beginning of 1977.

We are also presently working on the development of a pilot course and projects in the field of preventive medicine. This course would be part of a national effort on the part of AMSA to encourage the incorporation of the concept of health maintenence, as a basic responsibility of all individuals and health professionals, into medical education. Dr. Herb Abrams, Chairman of the Department of Family and Community Medicine and Ms. Shelley Clarfield, Administrator of the Health Manpower Distribution Demonstration Project are directing the planning of the course and related projects.

Once again, thank you for your support. We are looking forward to working with you this year.

Cheryl Pagel
Chairperson
Arizona AMSA Chapter

Editorial

OBSCENITY

WILLIAM B. McGRATH, M.D.

In his own lifetime a person recapitulates, condenses, summarizes the whole history of civilization. One goes from toilet training to table manners to the careful regulation of aggression in athletics and on to such larger institutions as marriage and religion and the law.

These institutions are the building blocks of civilization. They channelize or sublimate a brutality which is inherent in every basic drive or instinct. The bylaws, so to speak, make it possible for us to live in peace and cooperation. Without strict adherence to the rules we could not have anything from a game of bridge to a World Series or a motor trip in traffic.

An equation presents itself. When religion has strong control over our lives the law can be permissive. When the influence of religion is weak, then the law must become more rigid and repressive. Witness, for example, the strict regulation of personal behavior under atheistic dictatorships or in communism. The state becomes one's conscience.

Nudity is natural. So is complete lack of privacy. So is the absence of all civil rights.

Dress itself is conventional (assembly for a common purpose). Civilized people have always and everywhere worn clothing. Camouflage and climate are not the main determinants.

Garments are a symbolic protection, not against sexuality, but against the aggressive bestial elements in sex. This is important. Those of us who are conservative in such matters are accused of being somehow against sex and opposed to sexual freedom and honesty.

No! We resist the undermining of institutions which try to wall out the original brutality of sex. We resist regression and atavism, and rightly so.

This, in context, is not a moral issue. It pertains to the maturing of the individual in parallel with the progress of the race.

Now the first and most satisfying relationship of all is that of the child to its mother. A female child can prolong that relationship, staying close to the mother and becoming secure in her identity as a female. What about the male child? He has to be pushed away and later he has to push himself away in order to develop and be sure of his own maleness. There is no way that this can be realized without some hostility, some frustration and aggression. The female is not strongly admonished against peeping. She has nothing to lose. She does not have external genitalia or any need of physical potency or fear of its loss.

Oversimplifying, this is probably the source of the male's tendency to fear and therefore degrade the female. It is the origin of pornography, and pornography. We are frightened by the portrayal duced exclusively by men and for men. Again, we are not distrubed by the explicit sexuality of pronography. We are frightened by the portrayal of sexuality with little or no concern about love or compassion or dignity or commitment.

Only the human represses his bestiality by preferring the face-to-face position in intercourse and by going back to the breast in courting or foreplay.

Historically tenderness between the sexes is a relatively recent and incomplete achievement of humanity. Mass rape is still one of the spoils of war.

A few centuries ago the first laws on obscenity were not primarily concerned with sex. Someone had published a scurrilous attack on the titillating (excuse the word) questioning by the priest in the confessional. Reprisal was political; not against the sexual content but against the sedition of any attack on the government, which still comprised the church and state.

Our protest, to repeat, is against hostility and immature and uncivilized aggression.

All perversions are grounded in aggression. The voyeur or exhibitionist, for example, is invading privacy and taking masturbatory pleasure from a woman who would not willingly grant the use of her body for such a purpose if she were given a choice. The springtime fertility rite of panty raids is a more frivolous illustration of partially sublimated and adolescent sexual aggression. Not overlooked are contaminants of fetishism and transvestism.

From: 5051 N. 34th St., Phoenix 85018.

The woman's freedom of choice is the only absolute in the judgement of these matters. In a permissive society and with the law's ignoring of semantics, we say flatly that behaviour involving consenting adults is none of government's business. Yet we should take a longer look at that term, consenting. A young Vietnamese might "consent" to perform an unnatural act in order to keep her child from starvation. A prostitute might "consent" if the alternative is to lose her source of heroin or be beaten by her pimp. There are many subtle forms of barter and coercion which are hidden behind the concept of consenting adults.

So, too, with the term, victimless crimes. It is inherently contradictory. There is no crime without a victim; there is no victim without a crime.

Proponents of pornography argue about art and realism. Implicit in the definition of art, however, is that something is left to the viewer's imagination. Realism? No; the performers in a pornographic film are working for money and faking any mutual pleasure. A rape scene might seem realistic. But it is a still-life: It does not show the aftermath, the young woman's sterilizing surgery for gonorrheal abscesses of the ovaries.

The President's Commission on Obscenity in the Media contradicted the Commission on Violence. The portrayal of violence was deplored. Obscenity was said to have little or no influence on the behaviour of the viewer. Well, if the latter were true then one might scoff at the billions of dollars spent in advertising, trying to induce the viewer to purchase a product. If seductive portrayal has no influence on behavior, the whole advertising industry had as well go out of business.

There are reasons, deeper than hygiene, why we eat from plates and with utensils. We cannot revert to publicly killing and disemboweling animals and fighting over their bloody flesh. Is not pornography the slaughterhouse of sex? There is no law against the sexual behaviour in pornography; only against its public distribution and display.

All our taboos and customs have been necessary to prevent the dehumanizing of our communities and especially our children and our womenfolk. All of our cultural efforts are to hold under some kind of institutional control the fundamental savagery of man. We do condemn any concerted attack on institutions which suppress not sex but the primitive brutality of sex.

As with the indivdual so with the race: We are still on the threshold of civilization and we must not turn back.

Topics Of Current Medical Interest

HYPOCHONDRIACS ALWAYS IMPROVE FOR 3 MONTHS

Most of us go into medicine hoping to be helpful to other human beings. What is so frustrating about the hypochondriac patient is that you *cannot* be helpful . . . We fail to realize that the hypochondriac doesn't want to be rid of his symptoms. What he or she wants is a caring relationship.

Don't be misled by apparent early success: Everything works during the first 3 months—major or minor surgery, medications, placebo, hypnosis. The patient gratefully tells you that he is getting relief, for the first time.

At this moment, the experienced physician gracefully gets out . . . Very soon, the patient will have new symptoms or a return of the earlier symptoms.

Do take the hypochondriac off all medications—this is a very helpful step, as the patient may be taking from 6 to 10 prescriptions from former physicians. Clearly, these have not helped and should be phased out.

Don't repeat laboratory tests, radiologic examinations or invasive workups, if they have been found to be negative.

Do prescribe yourself or some other physician twice a month to let the patient ventilate her complaints. This may keep her from taking drugs . . . Sooner or later, this type of patient starts breaking appointments or tries to make other appointments, as she hears about another physician to visit.

Continued on Page 1044

Big Balanced Rock, Chiricahua Mountains, Arizona (approx. 1,000 tons)

- **Most Widely Prescribed**—Antivert is the most widely prescribed agent for the management of vertigo* associated with diseases affecting the vestibular system such as Menière's disease, labyrinthitis, and vestibular neuronitis.
- **Relief of Nausea and Vomiting**—Antivert/25 can relieve the nausea and vomiting often associated with vertigo.*
- **Dosage for Vertigo***—The usual adult dosage for Antivert/25 is one tablet t.i.d.

BRIEF SUMMARY OF PRESCRIBING INFORMATION

*INDICATIONS. Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: Management of nausea and vomiting and dizziness associated with motion sickness.

Possibly Effective: Management of vertigo associated with diseases affecting the vestibular system.

Final classification of the less than effective indications requires further investigation.

CONTRAINDICATIONS. Administration of Antivert (meclizine HCl) during pregnancy or to women who may become pregnant is contraindicated in view of the teratogenic effect of the drug in rats.

The administration of meclizine to pregnant rats during the 12-15 day of gestation has produced cleft palate in the offspring. Limited studies using doses of over 100 mg./kg./day in rabbits and 10 mg./kg./day in pigs and monkeys did not show cleft palate. Congeners of meclizine have caused cleft palate in species other than the rat.

Meclizine HCl is contraindicated in individuals who have shown a previous hypersensitivity to it.

WARNINGS. Since drowsiness may, on occasion, occur with use of this drug, patients should be warned of this possibility and cautioned against driving a car or operating dangerous machinery.

Usage in Children Clinical studies establishing safety and effectiveness in children have not been done; therefore, usage is not recommended in the pediatric age group.

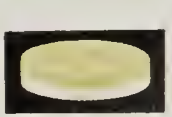
Usage in Pregnancy: See "Contraindications."

ADVERSE REACTIONS. Drowsiness, dry mouth and, on rare occasions, blurred vision have been reported.

More detailed professional information available on request.

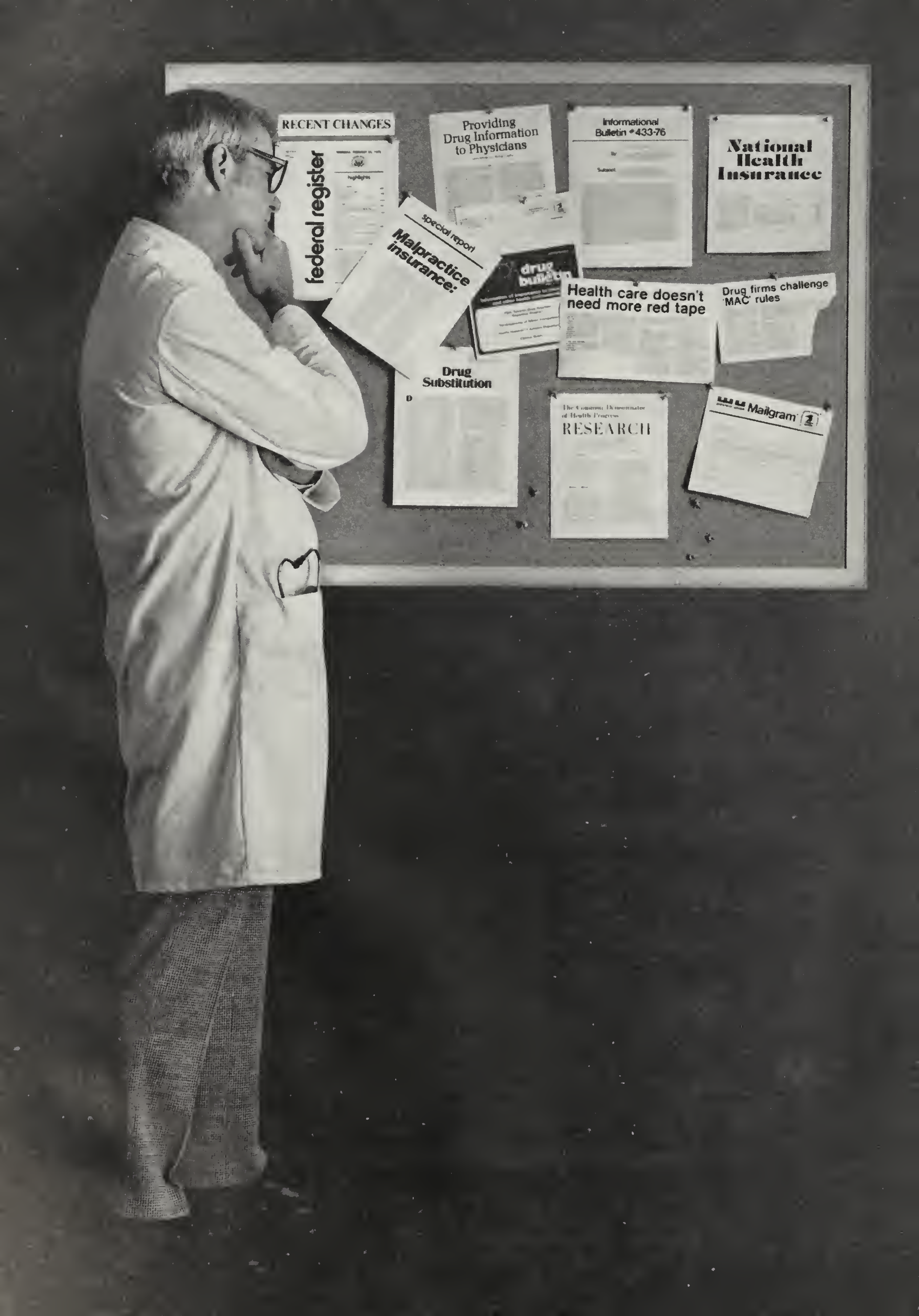
ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

Antivert®/25



(meclizine HCl) 25 mg. Tablets

for vertigo*


RECENT CHANGES
federal register
highlights
Providing Drug Information to Physicians
Informational Bulletin #433-76
Subject
National Health Insurance
special report
Malpractice insurance:
drug bulletin
Health care doesn't need more red tape
Drug firms challenge 'MAC' rules
Drug Substitution
The Common Denominator of Health Progress
RESEARCH
Mailgram

THERE ARE A LOT OF PEOPLE GETTING BETWEEN YOU AND YOUR PATIENT.

Medicine today is in the spotlight, subjected to all kinds of scrutiny. Your control over patient therapy is being monitored, judged and occasionally abrogated, sometimes by unknown third parties.

The worry is that in the wake of this focus, the relationship between you and your patient will be weakened, without offsetting benefits. Consider three examples:

Drug substitution In most states, pharmacy laws, regulations or professional custom stipulate that your non-generic prescriptions be filled with the precise products you prescribe. But in the last five years, a dozen or more State laws have been changed, permitting the pharmacist in most cases to select a product of the same generic drug to fill any prescription.

Ironically, this dilution of physician control has taken place against a background of growing evidence that purportedly equivalent drug products may be inequivalent, since neither present drug standards nor their enforcement are optimal. In fact, the FDA itself says it has not enforced the same standards for hundreds of "follow-on" products that it had applied to the original NDA approvals. Thus physician control over patient therapy is being eroded with a risk that patients may be exposed to drugs of uncertain quality.

The major advertised claim for substitution is reduced prescription prices for consumers. Yet no documentation of any significant savings has been produced.

MAC Maximum Allowable Cost, MAC for short, is a Federal regulation designed to cut the Government's drug bill by setting price ceilings for drugs dispensed to Medicare and Medicaid patients. Unless the prescriber certifies on the prescription that a particular product is medically necessary, the Government intends to pay only for the cost of the lowest-priced, purportedly-equivalent, generally-available product. The effect of the program may be that elderly and indigent patients will be restricted to products which someone in Washington believes are priced right. Practicing doctors will have little to say about administration of the program, since Government will have absolute authority to make its choices stick.

The drug lag The future of drug and device research depends upon a scientific and regulatory environment that encourages therapeutic innovations. The American pharmaceutical industry annually is spending more than $1 billion of its own funds and evaluating more than 1,200 investigational compounds in clinical research. Disease targets include cancer, atherosclerosis, viruses and central nervous system disorders, among others. But there is a major barrier to the flow of new drugs to your patients: The cost of the research is more than ten times what it was, per product, in 1962; and whereas governmental clearance of new drug applications took six months then, it commonly consumes two years now.

The FDA needs adequate time, of course, to consider data. But it is equally clear that the present approval process contributes to needless delay of needed therapy. That's why the increased efficiency of the drug approval process is vital to all our futures.

If these issues concern you, we suggest that you make your voice heard—among your colleagues and your representatives in State legislatures and in Washington.

It could make a difference in your practice tomorrow.

P·M·A

Pharmaceutical Manufacturers Association
1155 Fifteenth Street, N.W., Washington, D.C. 20005

Do keep your professional distance; *don't* get drawn into the hypochondriac's over-heated universe; *do* resist unctuous flattery, "Oh, doctor, I've heard so much about you!"

Remember: Any given physician is only a way station-stop in the hypochondriac's endless journey in search of the non-existent perfect healer.

—Rynearson, Robert: Stop Feeling Guilty In Treating Hypochondriacs. Med. Tribune 17:1-19 (Oct. 27) 1976.

PHYSICIANS CONTINUE TO LEAD IN PUBLIC CONFIDENCE POLLS

When a political candiate emerges the winner despite potent opposition from special-interest groups and the press, there is a standard way of describing his success:

"He had everybody against him but the people."

Today that could reliably be said of physicians. The polls show we're retaining our public esteem, regardless of onslaughts from various denizens of Capitol hill, regulatory agencies, Ralph Nader, Max W. Fine, and the *New York Times.*

According to a new survey by Louis Harris, the people view physicians more favorably than all other sources of their health care.

Topping the 15 categories surveyed are medical specialists, viewed favorably by 83% of the public and unfavorably by only 9%. Confidence in general practitioners has risen from 72% favorable-19% unfavorable in 1968 to 81%-16%.

Hospitals in general got a 70% favorable 27% unfavorable rating in the poll, which was conducted for the Federation of American Hospitals.

The cheering section for eventual federal takeover of care should take note that private, nonprofit hospitals received a 60% favorable-21% unfavorable rating, compared with 53%-20% for military and veterans' hospitals.

Although labor unions are among the glibbest pleaders of the "we demand better care for the people" line, union medical centers trail badly in public regard.

The Harris survey also shows that the public considers higher malpractice insurance premiums one of the five major causes of climbing medical and hospital costs—along with increases in nurses' salaries, physicians' fees, office overhead, and drug costs.

A flock of other surveys show the doctor is pretty darn popular.

Physicians were rated the most trustworthy of occupations in a survey by Chilton Research Services. In another survey, more than 8 of 10 people interviewed by Roper Reports said they are "very satisfied" or "fairly well satisfied" with both the quality and availability of their medical care. While a majority considered medical costs too high, 8 of 10 expressed satisfaction with their provisions for meeting those costs.

One good turn deserves another—so thank your patients for all these votes of confidence.

HIGHLIGHTS FROM THE STATE AUXILIARY

In the beginning, there were sixty-four Arizona doctors' wives who met in May, 1930, to organize the Woman's Auxiliary to the Arizona Medical Association. The state was geographically represented from Flagstaff to Nogales and Yuma to Safford with ten additional locations between. Forty-nine of the organizers were from the Phoenix group fromed in October, 1924, to provide "social assistance" to the doctors. That group joined the national auxiliary in 1931 and moved into health concerns with the state auxiliary.

The Arizona auxiliary has been continuously allied with the national structure which was organzied in May, 1922, when twenty-four women from eleven states met in St. Louis to persue the request to organize submitted to the A.M.A. House of Delegates by the four year old Texas auxiliary. Approval was granted with the observation that "the present plan does not appear to involve any financial or other obligation . . ." In 1923 the first annual meeting was held in San Francisco with a first year expense reported as "something near $20.00". Fifty years later the reported budget was $395,000.00, over $5 million had been given to the A.M.A. Education and Research Fund, and recruiting for the Health Fields prevailed.

The official Auxiliary Pledge was adopted in 1943, and the purpose has remained unswerving to assist in the advancement of medicine and public health. The Arizona Auxiliary avowed the cultivation of friendly relations and promotion of mutual understanding among physicians' families as well.

It has been the Medical Auxiliary pattern to establish program emphasis and leadership, then work together and with other groups in the community to achieve effective results.

It is noteworthy that the major continuous chairmanship is Legislation, reflecting the declaration at National's 50th Anniversary that it was uncertain whether #1 priority went to Membership or Legislation.

It might be suggested that trends in our country's national history are reflected in the ever-changing course and direction of Auxiliary activity. Currently, the Auxiliary name has been modified to encompass the mates of female physicians in the A.M.A.; further illustrating that, at 46 years and as many chairmanships later, the Medical Auxiliary remains eligible for new beginnings.

Letters to Editor

Editor Arizona Medicine
Dear Sir:

This is the story of my fight for financial and professional survival, but may be the fight of all other physicians.

For the most part of 23 years I have been the sole practitioner practicing in Eloy, Arizona. For those who do not know, Eloy is a city of over 7,000 people, drawing from over 15,000 in the metropolitan area, having an over abundance of welfare recipients, aged, indigents and farm laborers. The nearest hospital available, Hoemako, is in Casa Grande, 15 miles away. I have had hospital privileges at that hospital the entire time, and at no time have I ever had even a threat of a malpractice suit involving the hospital.

Six years ago I was refused coverage by Travelers Insurance Company and even the Arizona Medical Society said they could not find out why. For one year I could not even obtain malpractice insurance and practiced "bare," but finally obtained insurance through Signal-Imperial.

During the past year I watched the taxes, utilities and general cost of operation double. Malpractice insurance has quadrupled. I raised my fees to adjust to the increasing cost, but found many people going without medical care because they could not afford the fee. The county and medicare pay only half the regular office fee, $6.00 and $6.40 respectively. This does not cover overhead expense, and both the county and medicare have resisted any adjustment for increasing fees. Expenses had to be cut somehow.

The critical decision was finally made in July, 1975 when my brother informed me that in California the expense was so great that most doctors went "bare" and discovered to their pleasant surprise that this decreased their chances ten fold for law suit. When this was coupled with the notification from the State of California that Signal-Imperial had inadequate reserves and probably could not meet their obligations, I notified Hoemako Hospital Administration of my intent to drop my insurance. The hospital responded by quoting the following by-laws:

> "All staff members shall have and maintain insurance of a kind and amount required by the staff and approved by the governing body."

At no time had the hospital medical staff made any requirements as to insurance. The hospital stated that their carrier, St. Paul, would cancel its own insurance if 100% of the doctors did not carry malpractice insurance. When I contacted St. Paul I was informed that this was not the policy of that company. Indeed, one Phoenix hospital was insured by their company, and physicians desiring hospital privileges could receive same by a statement of financial responsibility.

In December, 1975, I attempted to get a clarification of the bylaws by inserting the words "or any other criteria" into the aforementioned statement in the bylaws. This was voted down, and the bylaws remained ambiguous. Nevertheless the Board of Trustees of Hoemako Hospital unilaterally stated that they would require a minimum of $250,000 malpractice insurance and suspended me. I responded by renewing my insurance, but filing suit to show cause why I could not practice without insurance.

On March 10, 1976, at an injunction hearing, the following letter was written and introduced in evidence, stating:

> TO WHOM IT MAY CONCERN
>
> This is to advise that the Medical Staff and Board of Trustees of Hoemako Hospital have historically for a number of years required that its Medical Staff members carried a minimum of $250,000 in professional malpractice insurance and that this requirement has been enforced.
>
> (s)
>
> Jay Wilson
> Vice President
> Board of Trustees
> Hoemako Hospital
>
> (s)
>
> R. C. Pogge, M.D.
> Chief of Staff
> Hoemako Hospital

This letter was a major factor in losing both the injunction and the Superior Court hearing. When it was pointed out that the staff had never made such a recommendation (as stated in the bylaws), the judge stated that clarification would entail a delay of at least three months. However, it was stipulated that if I was damaged by this letter, I could later sue for damages without prejudice. To rectify the oversight, the Board of Trustees requested the Medical Staff to uphold the action already taken. No physician would even make the motion, and it never came to vote.

In September, 1975, I hired a Physician's Assistant because I was unable to interest a physician in this area. Signal-Imperial "covered" him at no additional premium. When my insurance ran out, I applied to MICA and was placed in Class 5, although most doctors in Casa Grande doing the same work were in Class 2. In addition I was informed that I was assessed a surcharge of 100% for having a P.A. This brought the premium to approximately $50.00 per working day. This action was appealed, and I have not as yet heard what action will be taken on this appeal. If it is upheld, it means the end to the P.A. concept in Arizona.

An attempt was made for a "special action" by the Supreme Court since by this time I was feeling the pinch of the adverse publicity and the hospital's refusal to call me on my patients. The hospital fought the special action, stating there was no urgency in the matter. At the present time the case is in the Appellate Court, but a date for trial has not yet been set.

Anyone affected by the outcome of this case could enter the suit at this time as an "Amicus Curiae." This would include individuals also "going bare" and threatened with suspension of hospital privileges, specialty societies, or even the Arizona Medical Association itself. I realize that the tendency is not to "get involved." however, with MICA admitting that the premium will be doubled in the next three years, it would behoove anyone inclined to "go bare" to do so now. As you know, if any doctor does not continue with MICA, all the premiums paid will be lost. The minute premium payment ceases all pre-existing coverage is cancelled.

I am in the fight to the end. If MICA upholds the surcharge of 100% (or even if much less) I may be forced to "go bare" and accept the suspension. How long it will take, I do not know. I have appealed to my colleagues for help and the response, though meagre compared to the cost, is gratifying. Forty eight physicians have given over $2,000.00 in gifts of $10.00 to $300.00 in order to defray the legal expense. More is needed. But even more valuable would be those who would enter as an "Amicus Curiae," showing that the fight is not mine alone.

I still cannot accept that hospitals can dictate the kind and amount of insurance, at the same time saying that the doctor is an independent contractor and as such, the hospital cannot be responsible for his actions.

Sincerely yours,
H. Howard Holmes, M.D.

Dr. John W. Kennedy
Chairman-Publishing Committee
Arizona Medicine

Dear Doctor Kennedy:

I read, with interest, the article in the August, 1976 issue of Arizona Medicine, titled, "A Proposal: Some New, 'Old', Terms and a Scoring System for Irreversible Coma" by Gordon M. Greenblatt, M.D.

As with most attempts to simplify, this proposal instead creates some ambiguities which actually could be quite dangerous. I have seen patients that would be classified as "complete death" with 14 points make complete recoveries.

For example:

I. CNS, depressed, pupils dilated, respond to light, no response to painful external stimuli and or pathological reflexes. (2 points).

II. Cardiovascular, abnormal cardiac electrical activity and sustained chemically or by assisted or controlled respiration. (6 points).

III. Respiration, abnormal pattern of breathing, inadequate or adequate, may require mechanical assistance. (6 points).

The term "threatened death," of course this can apply to any of us, at any time, particularly when we are in a motor vehicle. The term "inevitable death," of course applies to all of us at all times. Adding the term "complete death" for death itself has not really improved my comprehension of our ultimate fate.

Fortunately, the title of the article is — a proposal — which, after evaluation, should be rejected.

Sincerely,
L. Philip Carter, M.D.

Dear Dr. Carter,

Thank you for your letter of October 19, 1976.

Your observation is a most valuable contribution to my proposal and perhaps a 14-point-scored patient could recover, as you say.

The points for the scoring system could be adjusted so that:

1) Cardiovascular; abnormal cardiac electrical activity, sustained chemically or by assisted or controlled respiration . . . equals 4 points.

2) Respiration; Abnormal pattern of breathing inadequate or adequate, may require mechanical assistance . . . equals 4 points.

3) Then: "Threatened Death" would be 4 to 10 points,
"Inevitable Death" would be 10 to 14 points,
"Complete Death" would be grater than 14 points.

As to your comments about the terms themselves, I only hope that very few active, healthy people are depressed to the point of applying such morbid terms to themselves.

Whatever "our ultimate fate" might be, I certainly hope it will never become a vegetative existence sustained by mechanical robots. If any scoring system can prevent this, I hope we continually upgrade this system, or propose a new one.

Sincerely,
Gordon M. Greenblatt, M.D.

Dear Sirs:

We are very pleased at the current leadership of the Arizona Medical Association and the recent poll of the membership regarding a unified membership issue. This should have been done many years ago and I think it speaks very well for the current leadership.

Additionally, we are pleased that a clear stance has been taken regarding the auto emission testing issue and again appland the leadership.

The full time members of our department, in view of the results of the recent poll on unified membership, would like to state our position on the issue. We feel that the organization should follow the mandate of the majority and drop the unified membership rule. If the organization does not follow the wishes of the majority, then we would be compelled not to join the organization.

We feel that ArMA has a vital role to play, one that will become even more important in the years to come. We respectfully request that our department's position be given consideration.

Sincerely,
Michael A. Platt, M.D.
Secretary,
Department of Internal Medicine
Pima County General Hospital

Dear Dr. Sattenspiel:

I have just finished reading your editorial entitled "A Paradox of Pressures" in this month's *Arizona Medicine*. I am very pleased that you have taken the time and effort to put a formal message in the *Arizona Medicine Journal* for the physicians of this state to see. We, in ophthalmology, have become quite active in the area of our specific problem with optometry. I believe that other specialties, such as orthopedics and psychiatry, need to become active in a similar fashion because this is not just a problem limited to the paramedical area of optometry, but it is a problem penetrating all areas of medicine. Our federal government is moving ahead with its plans for a national health insurance program and the paramedical groups are seizing upon this in an opportunistic fashion, attempting to gain any advantages possible. And why not? Who could blame them for asking for more, if they believe they can get it. The only problem is that the public will be the loser because of the confusion between who is the primary force of medical care.

While thanking you for your article, I must also urge you to continue moving our state medical society in ways that will help our subspecialties avoid the confusion that these paramedical specialties seek to foster. This should be a united effort of all of the sub-specialties and the state society can do much to help the situation.

Sincerely yours,
Lorin M. Swagel, M.D.

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona on Friday, October 1, 1976 convened at 6:58 p.m., Edward Sattenspiel, M.D., President and Chairman presiding.

SURVEY ON COMMITMENT LAW

The request from the Arizona Psychiatric Society that we conduct a survey of the membership soliciting their opinions on the commitment law (P. L. 1035) that went into effect on October 15, 1974 was discussed.

MEDICAL STUDENT'S ATTENDANCE AT AMERICAN MEDICAL ASSOCIATION, INC. CLINICAL MEETING

The Arizona Chapter of the American Medical Students Association's request that we provide them with funds equal to what we would spend to send one student to the American Medical Association, Inc. Clinical Meeting in Philadelphia was discussed. It is the intent of the students to send two people to the meeting, raising the difference between our contribution for one person and the cost of sending two people, from other sources.

IT WAS MOVED AND CARRIED TO APPROVE THE ABOVE REQUEST.

BLUE CROSS/BLUE SHIELD CONFLICT OF INTEREST

Recommendations for appointment

The question of the propriety of our making recommendations of physicians for possible election to either Board was discussed. It was pointed out that as of July 10, 1976 the Association no longer has any official tie to Arizona Blue Shield.

IT WAS MOVED AND CARRIED TO ADOPT THE POLICY OF NOT SUGGESTING NAMES FOR POSSIBLE ELECTION TO EITHER THE BLUE CROSS OR BLUE SHIELD BOARDS.

ArHA/ArMA COSPONSORSHIP

It was agreed to co-sponsor with the Arizona Hospital Association two programs (1) the medical staff in the modern hospital and (2) H. B. 2001 reporting requirements.

ArMA/MICA LOAN

Mr. Robinson explained that the $1,000,000.00 loan made by ArMA to MICA was about to be paid off except for the differential in the amount of interest paid by MICA (6%) and the amount to be paid by ArMA (7%). This difference would normally be paid at the rate of $100.00 per policy and would take several months. MICA has agreed to pay off the difference in full if approved by ArMA.

IT WAS MOVED AND CARRIED TO AUTHORIZE MICA TO PAY ArMA THE BALANCE OWING ON IT LOAN OBTAINED IN ORDER TO PURCHASE MICA'S CONTRIBUTION CERTIFICATE, INCLUDING INTEREST TO THE DATE OF PAYMENT.

1976 MALPRACTICE ASSESSMENT

Status Report at September 30, 1976

Mr. Robinson reported that as of September 30, 1976, one month after the initial billing, $120,900.00 had been collected from the 1976 Malpractice Assessment as follows.

	Total Billed	Total Paid	% of Total Billed
TOTAL	2165	1209	55.8

Other Response	Total Accounted for	% of Total Billed
19	1228	56.7

Requests for Exemptions

The following were exempted due to recent membership classification changes in accordance with the Bylaws Chapter IX, Section 4(b)

Louis C. Kossuth, M.D., Associate Member
Hugh C. Thompson, M.D., Over 70
Joseph A. Van Ham, M.D., Retired

"MEDICAL OPINION" TV SHOW

Mr. Robinson reviewed the desire of KOOL-TV to run the "Medical Opinion" TV Show on a weekly basis instead of monthly.

The Executive Committee strongly endorses this new format and asked that the P. R. Committee meet as soon as possible to establish topics and possible physician participants.

ARIZONA HEALTH SERVICE CORP.

Dr. Sattenspiel reported that our grant application has been approved in the amount of $101,349.00 and that he was going to suggest to the Board of Directors that the physician members of the Section on Rural and Migrant Health plus John F. Kahle, M.D. be designated as the Executive Committee to oversee the grant.

Meeting adjourned 9:01 p.m.

William E. Crisp, M.D.
Secretary

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, AZ on Saturday, October 2, 1976, a quorum being present, convened at 10:13 a.m., Edward Sattenspiel, M.D., president and chairman, presiding.

WELCOME

Dr. Sattenspiel welcomed the county medical society presidents and other guests.

ArMPAC

Dr. Langston reported on the current activities of ArMPAC indicating that the Board of Directors had recently met and made its decisions as to which candidates would be supported this year.

Confirmation

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF THOMAS F. MOORE M.D.

BOARD OF DIRECTORS

Southern District Director

IT WAS MOVED AND CARRIED TO APPOINT JOSEPH P. WHALEY M.D. TO FILL THE VACANCY OF ROBERT P. PURPURA M.D. TERM TO BE UNTIL THE NEXT REGULAR HOUSE OF DELEGATES MEETING IN APRIL 1977.

Opinion Poll — Unified Membership

The board reviewed the recent opinion polls regarding unified membership from members and non-members, the results of which were as follows:

OPINION POLL RESULTS — FROM MEMBERS
2487 QUESTIONAIRES WERE MAILED TO MEMBERS ON JULY 23, 1976
1086 or 43.67% OF THE QUESTIONAIRES WERE RETURNED BY CLOSE OF BUSINESS ON AUGUST 27, 1976

1. UNIFIED MEMBERSHIP	**Responses**	**Percentage**
A. I AM IN FAVOR OF THE ARIZONA MEDICAL ASSOCIATION MAINTAINING UNIFIED MEMBERSHIP WITH AMA	415	38.2%
B. I AM AGAINST THE ARIZONA MEDICAL ASSOCIATION MAINTAINING UNIFIED MEMBERSHIP WITH AMA	633	58.3%
C. I HAVE NO OPINION ON THIS ISSUE	38	3.5%
TOTALS	1,086	100.0%

OPINION POLL RESULTS — FROM NON-MEMBERS
1087 QUESTIONAIRES WERE MAILED TO NON-MEMBERS ON JULY 23, 1976
192 OF 17.66% OF THE QUESTIONAIRES WERE RETURNED BY CLOSE OF BUSINESS ON AUGUST 27 1976

1. UNIFIED MEMBERSHIP	**Responses**	**Percentage**
A. I AM IN FAVOR OF THE ARIZONA MEDICAL ASSOCIATION MAINTAINING UNIFIED MEMBERSHIP WITH AMA	33	17.2%
B. I AM AGAINST THE ARIZONA MEDICAL ASSOCIATION MAINTAINING UNIFIED MEMBERSHIP WITH AMA	151	78.6%
C. I HAVE NO OPINION ON THIS ISSUE	8	4.2%
TOTALS	192	100.0%

Following extensive discussion as to the meaning of the polls the following action was taken:

IT WAS MOVED AND CARRIED TO CALL A SPECIAL MEETING OF THE HOUSE OF DELEGATES FOR 9:00 a.m., SATURDAY, NOVEMBER 20, 1976 AT THE ASSOCIATION'S OFFICE FOR THE PURPOSE OF DISCUSSING AND VOTING ON THE FOLLOWING RESOLUTION:

Subject — Unified Membership
(Published in Arizona Medicine, Oct. 1976)

Survey of Optometrists

Discussion ensued regarding the request of the Arizona Ophthalmological Society that we conduct a survey of optometrists regarding the use of drugs.

IT WAS MOVED AND CARRIED TO APPROVE THE SURVEY TO BE CONDUCTED BY THIS ASSOCIATION AS REQUESTED BY THE ARIZONA OPHTHALMOLOGICAL SOCIETY.

BOMEX Appointments

It was announced that C. D. Buster M.D., whose term expires June 30, 1978, has resigned from the Board of Medical Examiners.

IT WAS MOVED AND CARRIED TO RECOMMEND, WITH THEIR APPROVAL, E. CHARLES BILL, M.D., COTTONWOOD AND GILBERT L. SECHRIST, M.D. OF FLAGSTAFF TO THE GOVERNOR AS CANDIDATES FOR APPOINTMENT TO FILL DR. BUSTER'S UNEXPIRED TERM.

EXECUTIVE COMMITTEE

Classification Changes Approved
Maricopa County Medical Society

(a) Jack A. Cannon, M.D. — Active to Service — Account Employment — One quarter dues Effective 1 1 77

(b) Robert K. Hilton, M.D. — Active Over 70 to Associate — Account Retirement — Dues Exempt — Effective 1 1 77

(c) Thomas C. Wilmoth, M.D. — Active Over 70 to Associate — Account Retirement — Dues Exempt — Effective 6 30 76

(d) Lewis S. Winter, M.D. — Active to Associate — Account Retirement — Dues Exempt — Effective 1 1 77

Pima County Medical Society

(a) Banks S. Baker, M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(b) Howard D. Cogswell, M.D. — Active to Associate — Account Retirement — Dues Exempt — Effective 1 1 77

(c) Donald F. Hill M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(d) Raymond F. Oyler, M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(e) Arthur J. Present, M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(f) Garfield P. Schnabel, M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(g) Harold F. Stolz, M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(h) Harry E. Thompson, M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(i) Hugh C. Thompson, M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(j) Clarence H. Kuhlman, M.D. — Active to 50 year Club — Dues Exempt — Effective 1 1 77

Pinal County Medical Society

(a) William P. Tucker, M.D. — Active to Active Over 70 — Account Age — Dues Exempt — Effective 1 1 77

(b) Glen H. Walker, M.D. — Active to Associate — Account Retirement — Dues Exempt — Effective 1 1 77

Yuma County Medical Society

(a) Wilbur D. Dice, M.D. — Active to Associate — Account Retirement — Dues Exempt — Effective 1 1 77

Request for dues exemption

IT WAS MOVED AND CARRIED TO APPROVE THE REQUEST OF THE COCONINO COUNTY MEDICAL SOCIETY'S REQUEST TO EXEMPT WILLIAM B. KENNEY M.D. FROM PAYING 1977 DUES.

FINANCE COMMITTEE

Loan to Mutual Insurance Company of Arizona

Mr. Robinson reported that as of September 3, 1976 the $1,000,000.00 loan to MICA had been repaid except for approximately $145,000.00 which is to be paid off in the next few days (NOTE: The loan was repaid in full on October 6, 1976).

Fiancial Statement for period ending 8 31 76

Dr. Clymer reviewed the subject statement for the Board as well as outlining the activities of the various Committees which are in the process of zero based budgeting. — RECEIVED

Narrative

The narrative of the financial statement which was prepared by Mr. Robinson at the request of the Board was received for information.

GOVERNMENTAL SERVICES COMMITTEE

State Health Coordinating Council

It was determined to recommend, upon their approval, the following to the Governor for possible appointment to the State Health Coordinating Council:

William J. Austin, M.D. — Flagstaff
Richard L. Dexter, M.D. — Tucson
Gilbert L. Sechrist, M.D. — Flagstaff
Wallace A. Reed, M.D. — Phoenix

Louisiana State Medical Society request re: P. L. 93-641

The letter from the subject organization requesting our support in challenging the constitutionality of P. L. 93-641 was reviewed and received for information.

PSRO and Federal health delivery systems

Dr. Reed reviewed the material from the AMA regarding the current status of the subject matter.

IT WAS MOVED AND CARRIED THAT THIS ASSOCIATION GO ON RECORD SUPPORTING THE CONCEPT OF PLACING ALL FEDERAL MEDICAL PROGRAMS UNDER THE JURISDICTION OF THE P.S.R.O. PROGRAM. AND THAT OUR POSITION ON THIS MATTER BE CONVEYED TO OUR CONGRESSIONAL DELEGATION AFTER THE ELECTION IN NOVEMBER, 1977.

Appointment Confirmations

IT WAS MOVED AND CARRIED TO CONFIRM THE APPONTMENTS OF DERRILL B. MANLEY, M.D. AS CHAIRMAN OF THE SECTION ON PLANNING AND WILLIAM M. DAVIS, M.D. AS CHAIRMAN OF THE SECTION ON MEDICAID.

GRIEVANCE COMMITTEE

Dr. Scott, Chairman of the Grievance Committee reviewed the deliberations of the committee at their June 19, 1976 meeting summarized as follows:

> The Committee received and reviewed the material dealing with the Department of Surgery at the University of Arizona College of Medicine which was referred to it by the Board of Directors.
>
> A great deal of material was made available to the Committee which includes the original request for an investigation, submitted to the Pima County Board of Mediation dated August, 1975, and the report of the Pima County Board of Mediation dated March 1976. It was noted that by action of the Board of Directors the Association has offered to act as a resource body for the Board of Regents, but said offer has been turned down.
>
> Considerable discussion ensued. It was pointed out that the questions raised in the Pima Report dealt primarily with what might constitute proper procedures within the Medical School for fairly hearing and deciding grievances brought by residents and others with respect to the Administration of the School. It was also pointed out that the power of the Arizona Medical Association's Grievance Committee as set forth in Chapter VII, Section IV(e) of the Bylaws, does not extend to the Administration of the Medical School.

Following considerable discussion the following action was taken:

IT WAS MOVED AND CARRIED THAT, AFTER A CAREFUL REVIEW OF THE REPORT OF THE PIMA COUNTY BOARD OF MEDIATION AND ALL OF THE MATERIALS SUBMITTED WITH IT, THAT THE ASSOCIATION TAKE NO ACTION AND MAKE NO FINDING AS TO THE MATTERS CONSIDERED THEREIN FOR THE REASON THAT THE JURISDICTION OF THOSE MATTERS APPEARS TO BE WITHIN THE PURVIEW OF THE ADMINISTRATION OF THE MEDICAL SCHOOL, THE UNIVERSITY OF ARIZONA, THE BOARD OF REGENTS AND THE COURTS.

HEALTH MANPOWER COMMITTEE

Confirmation of Appointments

IT WAS MOVED AND CARRIED TO CONFIRM THE FOLLOWING APPOINTMENTS TO THE HEALTH MANPOWER COMMITTEE:

Mr. David Beyer — Medical Student
Holley Faust, M.D — Housestaff

Arizona Health Service Corp. Grant

Dr. Sattenspiel reported on the fact that the Association's Foundation has received the H.E.W. Grant applied for by the Health Manpower Committee and approved by the Board of Directors on January 1, 1976. The Grant is a cost reimbursable grant in the amount of $101,319.00

Discussion ensued regarding the administration of the grant which resulted in the following action:

IT WAS MOVED AND CARRIED TO APPOINT THE PHYSICIAN MEMBERS OF THE SECTION ON RURAL AND MIGRANT HEALTH OF THE HEALTH MANPOWER COMMITTEE ALONG WITH JOHN F. KAHLE, M.D. TO BE THE EXECUTIVE COMMITTEE FOR THE GRANT WHICH WILL RESULT IN THE GROUP AS THE EXECUTIVE COMMITTEE.

ANDREW W. NICHOLS, M.D., CHAIRMAN
FREDERICK J. BRADY, M.D.
ALAN ENGELBERG, M.D.
HERBERT G. ERHART, JR., M.D
THOMAS W. JENSEN, M.D.
JOHN F. KAHLE, M.D.
ALEX KELTER, M.D.
LOUIS C. KOSSUTH, M.D.
JULIAN C. LENTZ, JR., M.D.
JOHN G. LINGENFELTER, M.D.
JOHN B. MILLER, M.D.
HOWARD NORMAN, M.D.
OTTO SEIBER, M.D.
MARK J. WESTERVELT, M.D.

MALPRACTICE INSURANCE CRISIS COMMITTEE

Solicitation of Voluntary Contributions

It was reported that the solicitation of non-member physicians, including doctors of osteopathy, resulted in $1,700.00 from the following:

Earl E. Bleak, M.D.
Joseph J. Bonanao, M.D.
Cooley Butler, II, M.D
Joseph R. Demosthenes, M.D
Joseph S. Gimbel, M.D
Theodore C. Harper, M.D
Sidney Hirsch, M.D
Donald J. Holmes, M.D
Douglas W. Huestis, M.D
J. Howard Johnson, M.D.
Arthur P. Klotz, M.D
James W. Lohman, M.D.
Daniel C. MacDougall, M.D.
Robert C. Millam, M.D.
Robert B. Patterson, M.D.
William W. Pretlow, M.D.
Maurice B. Ruland, M.D.
Leonard B. Schultz, M.D.
Elsa J. Sell, M.D.
Russell D. Simbari, M.D.
Ronald W. Vinyard, M.D.
Emory D. Warner, M.D.

John A. Bamberl, D.O.
Robert S. Barbosa, D.O.
James F. Brown, D.O.
David J. Chakmakian, D.O
Maury H. Cohen, D.O
H. W. Dunford, D.O.
Spencer D. Ellsworth, D.O
Charles R. Ganzer, D.O
Michael J. Gander, D.O
Roger L. Gaines, D.O
Leroy R. [illegible], D.O
Philip H. Lewis, D.O
Robert G. Maxfield, D.O
Royce W. Maxfield, D.O
Edward P. McDermott, D.O
Robert Ostwinkle, D.O.
Leonard Parfitt, D.O
Don F. Ranney, D.O
Ronald B. H. Sandler, D.O.
Berton Siegel, D.O.
William L. Smith, D.O.
Donald L. Starkey, D.O
Paul M. Steingard, D.O.
Donald E. Till, D.O
Donald S. Welch, D.O

1976 Assessment

It was reported that as of September 30, 1976, $120,900.00 has been received for the special assessment billing which was mailed on September 1, 1976.

MATERIAL AND CHILD HEALTH CARE COMMITTEE

Confirmations of Appointments

IT WAS MOVED AND CARRIED TO CONFIRM THE FOLLOWING APPOINTMENTS TO THE COMMITTEE:

WAYNE BECK, M.D — HOUSESTAFF SECTION
MS. KATHY REED — MEDICAL STUDENT

MEDICAL ECONOMICS COMMITTEE

Confirmation of Appointment

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF RICHARD W. SWITZER, M.D. AS CHAIRMAN OF THE MEDICAL ECONOMICS COMMITTEE.

PROFESSIONAL COMMITTEE

Confirmation of Appointment

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF WILLIAM C. BRAINARD, M.D. AS CHAIRMAN OF THE SECTION ON ATHLETIC MEDICINE

Statement on Marihuana

Dr. Linkner reviewed a proposed statement on marihuana requested by the Young Lawyers Section of the Arizona Bar Association which was followed by considerable discussion and comment.

IT WAS MOVED AND CARRIED TO CORRESPOND WITH THE YOUNG LAWYERS SECTION OF THE ARIZONA BAR ASSOCIATION STATING THAT THIS ASSOCIATION STANDS READY TO PROVIDE SCIEN

TIFIC INFORMATION AT ANY TIME ON ANY MEDICAL SUBJECT WHEN REQUESTED.

SCIENTIFIC ASSEMBLY COMMITTEE

Confirmation of Appointment

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF WAYNE BECK, M.D., HOUSESTAFF MEMBER, AS A MEMBER OF THIS COMMITTEE.

Steak Fry

IT WAS MOVED AND CARRIED THAT ONLY TWO FREE TICKETS BE PROVIDED TO EACH FIRM EXHIBITING AT THE ANNUAL MEETING; AND FURTHER, THAT EACH TICKET, BOTH COMPLIMENTARY AND SOLD, WOULD PROVIDE FOR TWO FREE DRINKS.

OTHER BUSINESS

Confirmation of Appointments

IT WAS MOVED AND CARRIED TO CONFIRM THE FOLLOWING MEDICAL STUDENT APPOINTMENTS.

MR. DOUG OUTCALT — LEGISLATIVE COMMITTEE

MR. ROB WILSON — MEDICAL EDUCATION COMMITTEE

Meeting adjourned 12:49 p.m.

William E. Crisp, M.D.
Secretary

ARIZONA

Antituberculosis Association Organized.—On May 22, the Arizona Association for the Study and Prevention of Tuberculosis, in affiliation with the national association, was organized with the following officers: President, Dr. John W. Foss, Phoenix; vice-presidents, K. C. Babcock, Tucson; Dr. John E. Bacon, Tombstone, and Rev. J. E. Critchfield, Phoenix; secretary, Dr. John W. Flinn, Prescott; treasurer, Lloyd Christy, Phoenix, and consulting medical board, Drs. Otto E. Plath, Phoenix; Harry T. Southworth, Prescott; W. Vincent Whitmore, Tucson, and William D. Cutter, Bisbee.

State Society Meeting.—The eighteenth annual meeting of the Arizona Medical Association was held in Prescott, May 18-20, and the following resolutions were unanimously adopted:

Stimulated by the clear and comprehensive study on preventive medicine given this morning by Dr. W. Jarvis Barlow, of Los Angeles, we, the committee appointed to draw up resolutions embodying his suggestions, submit the following:

1. *Resolved*, That the Arizona Medical Association recommends the formation of an Arizona Public Health Association, whose object shall be a study of public health problems, the education of the public and cooperation with the various boards of health of Arizona.

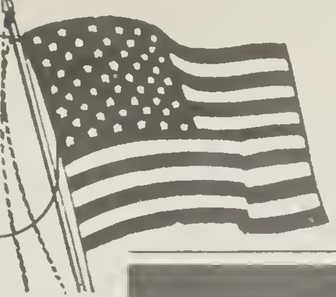
2. *Resolved*, That the Arizona Medical Association recommends the compulsory registration of all contagious and infectious diseases, including tuberculosis and compulsory vaccination of school children and systematic revaccination.

3. *Resolved* That the Arizona Medical Association recommends that such legal steps be taken as will insure thorough inspection and regulation of the milk supply.

4. *Resolved*, That the Arizona Medical Association appoint a committee of five to report through its official organ, the *Practitioner*, and to the secretaries of the county societies, such information as they can obtain regarding public health legislation and organization and to formulate a definite program to lay before the constitutional convention of Arizona, and that on the recommendation of this committee a special meeting of the Arizona Medical Association be called to adopt such a program.

The following officers were elected: President, Dr. Robert N. Looney, Prescott; vice-presidents, Drs. John W. Foss, Phoenix; William D. Cutter, Bisbee; and Edward S. Godfrey, Tucson; secretary, Dr. John W. Flinn, Prescott; treasurer, Dr. Enoch B. Ketcherside, Yuma, and essayist, Dr. W. Warner Watkins, Phoenix. Phoenix was selected as the place of meeting for 1910.

(Excerpt-Journal Amer. Med. Assn.-1909)



HISTORY OF THE WALTER O. BOSWELL MEMORIAL HOSPITAL

MRS. C. C. LOUDON
MRS. LUELLA LEISY

INTRODUCTION

A history must necessarily be constructed on a framework of dates and facts. Our hope is that the reader will see beyond the statistics to the countless hours spend and to the outstanding devotion to a dream—a dream of a hospital in the Sun City area.

Boswell is the outgrowth of that dream, generated by the needs of a rapidly growing suburban community with a heavy concentration of older residents with special needs.

Now the dream is a reality—a fine hospital standing ready, when illness or accident strikes, to stretch forth its facilities for relief and comfort to all.

1965

Sun City, only five years old, was growing at a phenomenal rate. It became more and more obvious that it was time to consider the feasibility of building a hospital to care for the many health needs of the rapidly increasing number of residents of retirement age. At that time, it was necessary for any resident with a major health problem to travel to a medical clinic or hospital in Phoenix. Since there was no ambulance service in Sun City, the time consumed in transporting the victim of a heart attack or stroke to an intensive care facility was, at best, dangerous and sometimes fatal.

This acute need weighed heavily on the minds of many concerned residents and officials of the Del E. Webb Development Co. Among these were Burt Welch, one of the concerned residents and Tom Austin, representing the Webb Company as Activity Coordinator for Sun City. These men were primarily instrumental in instituting an in-depth study on the feasibility of planning, building, furnishing, equipping and staffing a first rate hospital for the Sun City area. In connection with study, the Del E. Webb Development Co. conducted an opinion survey of Sun City residents. The response was highly favorable.

During 1966, Mr. Welch and Mr. Austin, who had been designated by the Webb Company as Director of Hospital Planning, conducted extensive research relative to the need for a hospital. They interviewed many physicians in the Phoenix area with regard to ways of attracting physicians, technicians and other ancillary personnel to a hospital in this area and how to best contact such people

As the result of their study, Mr. Welch and Mr. Austin concluded that the time and conditions justified formation of a Corporation for the purpose of working out a definite plan. To this end, they arranged a meeting with the following: John Meeker, President of the Del E. Webb Development Co.; W. A. Warmer, also of the Webb Company; Michael Bross, President of the home Owner's Association of Sun City; Luella Leisy, a pioneer resident; and Don Middleton, legal counsel for the Webb Company. On *AUGUST 16*, this small group founded THE SUN CITY COMMUNITY HOSPITAL, INCORPORATED.

For the balance of the year and for several months thereafter, the Board of Directors was involved in many meetings regarding such items as proposed architectural renderings and various fund raising projects. Medical clinics and nursing shcools in key institutions throughout the United States were contacted. Plans were finalized for an initial presentation to the Maricopa County Health Facilities Planning Council. Following this first presentation, the continuing rapid growth of Sun City presented further problems and gave rise to some misgivings as to the adequacy of the original plan for a 61 bed, one-story facility to be located on a five acre plot at 101st Avenue and Coggins Drive.

1967

On *FEBRUARY 17* an unexpected and gratifying development caused an abrupt and radical change in plans. On that date, James G. Boswell II, President of the J. G. Boswell Company and the secretary of the J. G. Boswell Foundation of California, accompanied Mr. Meeker to the regular monthly meeting of the Board of Directors for the purpose of advising the Board of the interest of the Boswell Foundation in the proposed hospital. The Boswell Foundation offered to grant $1,200,000 to the project, provided that the plan be expanded to a minimum of 100 beds; that the facilities be the best obtainable; that the proposed facility serve the entire surrounding community; and that the hospital be named the Walter O. Boswell Memorial Hospital in honor of Colonel Walter O. Boswell. Colonel Boswell was one of three brothers instrumental in developing a vast agricultural empire in this area, including land now occupied by Sun City. The Board of Directors unanimously accepted the offer. This development changed the concept from a small-town, one-story type hospital to a potentially large urban health facility, destined to become one of the largest and best in Arizona.

The generous gift of the Boswell Foundation was soon followed by the surprise announcement of the Webb firm of their intention to expand the Sun City development beyond its present limits to the area north of Grand Avenue. With this expansion in mind, the Development Company offered to donate 10.6 acres of land between Thunderbird Boulevard and Santa Fe Drive, a location more centrally located for future residents. Further, the Del E. Webb Corporation offered to build the type facility outlined in the Boswell Foundation offer at no profit to the Corporation.

1968

In *JANUARY*, William A. Chapman, Jr., former President of Town Hall Center, the organization of residents operating the recreational facilities in Sun City, was designated to assist in planning the hospital. On *FEBRUARY 1*, Mr. Chapman was elected as a member of the Corporation and of the Board of Directors. With the escalation of activities and additional responsibilities, the number of Corporation members and members of the Board of Directors was expanded.

The Board met with representatives of the Maricopa County Health Facilities Planning Council. That meeting lead to the selection by the Council and the Hospital Board of a JOINT COMMITTEE for the stated purpose: "to explore ways in which the Sun City Community Hospital Corporation and the Health Facilities Planning Council might work toward development of a HOSPITAL-MEDICAL care program in Sun City which would complement and supplement existing health resources in Maricopa County." Board members Michael Bross, William Chapman and Owen Childress with legal counsel, Don Middleton, were named to this committee to work on the joint effort as outlined.

In *MARCH*, Landon B. Atkins and Gerald W. McCarty were elected as additional members of the Board of Directors. At a meeting of the Board during this month, a representative of Haney Associates together with an advisory board composed of Bayard Horton, M.D., Leonard Larson, M.D., Charles Watkins, M.D., Oscar Friske, M.D. and Edgar Deissler, M.D. participated in discussions of plans and progress with the Board.

On *APRIL 5* the Hospital site was dedicated at a ceremony at which Del E. Webb and James Boswell were the featured speakers. The property on which the Hospital is built together with additional space for future expansion was officially transferred by the Del E. Webb Development Co. to the Sun City Hospital, Inc.

During *APRIL*, President Chapman, on behalf of the Corporation, signed rent-free leases for office space to be used by the Director of Planning and Fund Raising and for later use by the Hospital staff until completion of the building and opening of the Hospital. Arrangements were made for the loan of office equipment and for insurance on this equipment.

The Boswell Memorial Hospital Auxiliary, with 31 Founders and 127 Charter Members, was organized with Margaret Newby as the first President. Among the first projects of the Auxiliary was the serving of coffee and doughnuts at the kick-off meetings for the first fund drive on *APRIL 16 and 17*.

In *MAY*, Arizona granted the Sun City Community Hospital, Inc., tax-free status.

During early planning years, there were extensive expressed misgivings as to whether adequate medical and supportive professional personnel could be attracted to a facility such as that proposed and in this location. This was, in part, based on an Arizona law requiring certain qualification examinations before a physician moving from another state could be licensed to practice in Arizona. Legislative action changed the law and the Medical Committee soon became busy reviewing applications and interviewing applicants for the Medical Staff.

In the spring of this year, Landon Atkins was named General Campaign Manager for the first intensive campaign for funds to build the Hospital.

By *JULY* the campaign total had reached $179,000 and Merle Dennis was named Vice General Chairman in preparation for renewed fund raising efforts.

In *JUNE* the bylaws of the Boswell Memorial Hospital Auxiliary were approved.

In *NOVEMBER*, Alan Mann, the second Treasurer of the Corporation died.

1969

On *JANUARY 6*, the Auxiliary opened the first RESIDENTS' GALLERY in space made available in the Sun Bowl Plaza Shopping Center of Sun City by the Del E. Webb Development Co. This project was destined to become the most profitable enterprise of the Auxiliary with hundreds of hours donated by members both prior to and after the opening of the Hospital.

On *JANUARY 24* ground was broken for the building of the Hospital at an official ceremony. It was a rainy day with a chill wind but none-the-less, the first shovels full of dirt were turned by Del E. Webb and William W. Boswell, Sr., only surviving brother of the three who organized the J. G. Boswell Company. Thereafter turns at the shovel were taken by: Merle Dennis, Spring Fund Drive Chairman; Edward H. "Ted" Brainard, Fall Chairman; and Carl H. Anderson, February Drive Chairman.

Hospital assets had, by this time, increased to approximately one million dollars plus a grant of $500,000 from the Del E. Webb Foundation; a gift of $25,000 from Mrs. William Boswell and a gift of $50,000 from Mrs. Kay Evarts.

In *MARCH*, Eugene Coffman, Jr. was named to the Board to assume the duties of Treasurer.

In *JULY*, the construction loan was consummated with the First National Bank of Arizona and construction of the power house was added to the contract, bringing the total estimated cost to $4,507,148.

With construction under way, attention was turned to a Fall Fund Raising Campaign that would put us over the $1 milion total for the local drive. With the dedicated help of hundreds of local residents, and with a special grant of $50,000 from the Kresge Foundation, the goal was reached on *DECEMBER 16*. Sun City's population had reached approximately 12,000. An analysis of the drive disclosed slightly more than 52% participation with almost 99% of the pledges being paid. By the end of the year, approximately $800,000 had been collected.

During *DECEMBER*, a special Corporation meeting approved amendments to the Articles of Incorporation and the bylaws, changing the annual Corporation meeting to the first Tuesday in October.

1970

In *JANUARY*, the Board authorized the purchase of beds and other furnishings for the Hospital.

The following were appointed to a Medical Staff Organizing Committee: Bayard Horton, M.D.; Leonard Larson, M.D.; Charles Watkins, M.D.; Walter Sittler, M.D.; and E. B. Kuhe, M.D.

Arrangements were made for the temporary use of the second floor of a building in Peoria, Arizona to provide needed office space for Hospital personnel involved in recruiting, planning, etc. This space was used until the office area of the Hospital became available, September 14.

A group life insurance plan for employees became effective on January 1.

By *FEBRUARY*, 284 applications for employment had been received.

On *MARCH 1*, Marian Clark, R.N.M., Director-Nursing Services, began recruiting and organizing the nursing staff of the Hospital.

In *APRIL*, the Board received a construction progress report from the Webb Corporation showing substantial savings under estimated costs thus far. Ultimately, sufficient savings were effected to pay for all landscaping costs.

A utilities agreement for underground gas and electric lines was executed with the Arizona Public Service Company.

A draft of BYLAWS, RULES AND REGULATIONS OF THE MEDICAL, DENTAL AND RESEARCH STAFF, prepared by the Medical Staff Organizing Committee as a working document to guide in the selection of staff members, was approved by Resolution of the Board.

The Board approved the recommendation of the Medical Staff Organizing Committee in naming the following as the first members of the Boswell Memorial Hospital Medical Staff:

Richard Belgrade, M.D.
James Fuzzell, M.D.
Joseph Marcarelli, M.D.
William Minturn, M.D.
A. L. Rhoades, M.D.
Daniel Shanahan, M.D
Donald Tobias, M.D.
Jack Cannon, M.D.
Floyd Gindhart, M.D.
Baroukh Marzouk, M.D.
Jerome Reichenberger, M.D.
Leland Sargeant, M.D.
Walter Sittler, M.D.
Robert Waldman, M.D.

In *MAY*, the following were added to the Staff: Oscar W. Friske, M.D.; Robert Stump, M.D.; Edgar Dessler, M.D.; and McDonald Wood, M.D. Jack Cannon, M.D. was appointed Head of the Department of Surgery.

On *AUGUST* 13, Bayard Horton, M.D. was elected to membership on the Board of Directors and was appointed Chairman of the Medical Committee. Walter Sittler, M.D. was appointed Chief of Staff and Chief of Medicine of the Medical Staff.

Also in *AUGUST*, steps were taken to develop and inaugurate a Health Maintenance Program to supplement our in-patient program and to provide an organized plan for ambulatory care, extending health-care services into the community and the homes of residents.

The Administrator announced the filling of all necessary department head vacancies in our personnel complement.

Two classes for the training of Nursing Assistants were being conducted, utilizing the facilities of John C. Lincoln Hospital for clinical training of the students.

In *SEPTEMBER*, the following selections were made by the Medical Staff: Irving Pallin, M.D. as head of Anesthesiology; Robert Waldman, M.D. as head of Diagnostic Services; and Oscar Friskie, M.D. as head of General Practice.

Hospital offices and the entire staff moved from temporary quarters in Peoria to the nearly finished Hospital building.

The annual Corporation meeting on *OCTOBER* 6 re-elected all Corporation officers and aded Carl H. Anderson, Merle Dennis, B. R. Van Buskirk and Virgil Williams to the membership of the Corporation.

In *OCTOBER*, just a few weeks before the completion and dedication of the Hospital, word was received of the death of William W. Boswell, affectionately known to the Hospital staff as "Mister Bill," a member of the pioneer Boswell family. Mr. Boswell had been deeply interested in the building progress and had impatiently looked forward to the completion of the Hospital.

On *NOVEMBER* 6, the great dream was fulfilled with the dedication of the Walter O. Boswell Memorial Hospital. The ceremonies were held on the parking lot west of the building with over 5,000 guests in attendance. Board President, William A. Chapman, presided; Brigadier General James O. Boswell, son of the man for whom the facility is named, turned over a symbolic key to Administrator, W. Austin Turner; and Mrs. Carol Cramer, daughter of Burt Welch, the first Corporation President, sang a solo. Others who were present and took part in the ceremonies were: Bayard Horton, M.D., Emeritus Staff of Mayo Clinic, member of the Boswell Board and Chairman of the Medical Staff Committee; Del E. Webb, head of the Corporation bearing his name and developer of Sun City; and Henry Raymond, Board member and Manager of the Boswell Company of Arizona Projects. Frederick N. Elliott, M.D., Assistant Director of the American Hospital Association was the key note speaker. Tours of the new facility followed and continued for several days to accommodate those who were interested.

On *NOVEMBER 16*, the first five patients were admitted and Boswell Hospital was in operation with full 24 hour Emergency Room coverage. Walter Sittler, M.D., Chief of Staff, was on hand to greet the first inpatient, Mr. Stewart Beecher.

During the first fifteen days of Hospital operation, [illegible] five patients were admitted with Medicare accounting for [illegible] to 70% of the revenue.

1971

In *APRIL*, the Hospital Board of Directors approved the Articles of Incorporation for the Sun City Hospital Foundation and, by Resolution, provided for a line of credit and capital funds for establishment of the Foundation which will be the responsibility of a separate Board of Directors.

Boswell was approved by the Arizona State Board of Nursing for clinical training for student nurses.

The first of a series of training classes for supervisory development was held under the leadership of Board member, John R. Mead.

A program of Community Health Seminars, co-sponsored by the Hospital and the Sun City Community Association, was instituted.

In *MAY*, Boswell Memorial Hospital was accepted for membership in the American Hospital Association and Administrator W. Austin Turner was elected to the Board of Directors of the Arizona Hospital Association.

In *JULY*, the Board accepted the gift of a mobile-image intensifier from the Boswell Hospital Auxiliary, another outstanding contribution from this dedicated group.

The B-1-9 Lions Club contributed substantially toward the purchase of a Fundus Camera for use in detecting eye damage.

Speech Therapy service was established on an on-call basis.

In *AUGUST*, the Board was advised that, because of the price and wage freeze imposed by the President of the United States, all pay raises and rate adjustments were frozen for an indefinite period.

In *SEPTEMBER*, the Telephone Information and Reassurance Service, under the direction of the Home Service Department and staffed by Volunteer personnel, was inaugurated.

1972

In *JANUARY*, the cost of completion of the shelled-in fourth and fifth floors was estimated by the Webb Company at $1,010,000 plus $215,000 for equipment.

Foundation President William B. Gay reported receipt of pledges amounting to $272,000 with the assurance of an additional pledge of $15,000.

In *FEBRUARY*, the first transfer of funds from the Foundation to the Hospital was effected.

The Administrator reported that the average patient occupancy per day was running 92 as compared with 82.5 in January. These must be accommodated within a bed complement of 95.

Construction in the completion of the fourth and fifth floors was reported as being ahead of schedule with "substantial completion" expected in June. The projected opening date for the 5 West unit was set for the first week in October.

In *MAY*, the Foundation reported that the Fund Drive goal of $1 million had been reached. Cyril Rogers was hired as Executive Director of the Foundation to be effective July 1.

In *JULY*, an expensive Cavitron Kelman Phaco Emulsifier, equipment used in the removal of cataracts, was personally purchased by Ronald W. Barnet, M.D., and placed for use in the Boswell Surgical Suite.

The Administrator reported an average daily in-patient occupancy of 72 as compared with 55 in 1971, and an active Home Health Care Service patient enrollment of 25.

Auxiliary membership had increased to 711.

In *NOVEMBER*, the Administrator announced that patient care days for October totaled 2,770 and that five elective surgery patient reservations had to be cancelled due to overcrowding.

Medical Staff officers elected for the coming year were: C. Thomas Read, M.D., CHief of Staff; George L. Cannon, M.D., Vice Chief of Staff; and Kenneth H. Huey, M.D., Secretary-Treasurer.

The association of the "Meals-on-Wheels" program with the Hospital was approved, including approval of the bylaws of that organization.

DECEMBER—To date, the Foundation had donated over $567,000 to the Hospital.

The Board was advised of a bequest to the Hospital of approximately $50,000 from the estate of Robert A. Steffens.

The Home Care Program was certified for Medicare reimbursement.

Luella Leisy, one of the original incorporators, was made a life-time Honorary Member of the Board of Directors, in recognition of her long and faithful service as Secretary of the Corporation.

1973

In *FEBRUARY*, in recognition of the excellent progress in construction and operation of the Hospital, the J. G. Boswell Foundation made a grant of $1 million. Half of this grant was made available immeidately, with the stipulation that these funds be used to reduce the Corporation indebtedness.

The Webb Company's final construction billing for completion of the fourth and fifth floors in the amount of $1,035,508 disclosed a savings of $35,133 under the original estimate.

Long range financing for additional expansion by a bond issue was being explored.

In *AUGUST*, the first issue of the Boswell Hospital Pharmacy Newsletter, containing information about new products, the problems with such products, and other information necessary to keep physicians and nurses currently informed, was distributed.

Continuing their phenomenal program of donation of funds to the Hospital, the Auxiliary donated $22,700 for the purchase of Pacemaker clinical equipment, two electrocardio recorders and supplies, an Osmometer, and hot food delivery system equipment. This gift brought the total contributions by the Auxiliary to $99,657.

The Board adopted a Resolution setting forth standards of conduct and requiring Board members and the Administrator to disclose all factors that could result in a conflict of interest.

At the request of the Foundation President, an ad hoc committee composed of the Chairman of the Finance Committee, the Chairman of the Executive Committee, and the Treasurer, met with representatives of the Foundation to review financial problems confronting the Corporation.

Merle Dennis, who had been very active in various fund drives, resigned as a member of the Corporation and was given special recognition.

Gerald McCarty announced his decision to resign as Corporation Treasurer.

On *OCTOBER 2*, the annual Corporation meeting adopted certain amendments to the Articles of Incorporation.

C. Thomas Read, M.D., Chief of Staff, reported that 49 new physicians had been approved for Staff privileges during the year, 17 of whom have offices in Sun City. New services instituted at the Hospital included Endoscopic and Pulmonary Disease Service.

An architectural rendering of Boswell Memorial Hospital, showing the proposed expansion including the two additional towers, was the object of a surprise presentation to Board President Chapman by the Administrator on behalf of the "Boswell Family."

At the *OCTOBER* Board meeting, the following officers were elected for the coming year: President, William A. Chapman; Vice President, Landon B. Atkins; Secretary, John R. Mead; and Treasurer, Eric W. Walley. Facius Davis was elected to the Assistant Treasurer position and Gerald McCarty was made a Lifetime Honorary member of the Board of Directors.

Newly elected Medical Staff officers for 1974 were: George Cannon, M.D., Chief of Staff; Kenneth Huey, M.D., Vice Chief of Staff; Alice Palmer, M.D., Secretary-Treasurer; and Joseph B. Priestly, Jr., M.D. to the newly created position of Member-at-Large.

1974

In *FEBRUARY*, the Foundation President presented the hospital Board with a "package" compiled by the Foundation for presentation to newcomers to Sun City. The "package" contained information about the Hospital, the Foundation, the Speaker's Bureau, the Home Care Program, the Boswell ID card, the Pooled Income Fund, memorials, etc. He also reported that the Pooled Income Fund contained $120,000 as of the end of 1973.

In *MARCH*, the first blood-pressure clinic, co-sponsored by Boswell under the guidance of the Community Relations Committee, proved to be particularly successful. An estimated 2,400 residents were screened and some 150 were found to be hypertensive.

In *APRIL*, the Administrator announced that the Hospital had been awarded a full two year accreditation with a minimum of recommendations for improvement. This is our second consecutive full two year accreditation, resulting from the only two inspections conducted since opening.

In *JUNE*, the Board authorized the filing of applications for approval to expand the Hospital with both the Health Planning Council and the Arizona Department of Health.

The records showed that during a six month period, 53 patients were transferred to other hospitals because of the need for more extensive neurological evaluation and more extensive cardiac observation or surgery.

Substantial increases in the cost of supplies and services continue to be a problem. Salaries and wages, however, remain under budget even though cost-of-living increases have been granted in accord with Hospital policy.

The new Thunderbird Residents' Gallery opened and was an immediate success. Auxiliary membership totaled 924, not including 45 active Junior Volunteers.

The Auxiliary voted to donate another $23,380 for the purchase of a Bi-Plane Film Changer.

The Executive Committee recommended approval of a Residency Program in cooperation with the University of Arizona, School of Medicine, on a student-by-student basis for the coming year.

The Auxiliary reported having purchased equipment for the Hospital, totaling $123,077 to date, and having grown in membership from 31 Founders to 992 members as of September 1974.

Contributions to the Hospital during the seven year period consisted of: $2,700,000 by the J. G. Boswell Foundation; land valued at $265,000 at appraisal date from the Del E. Webb Development Co.; and $2,233,659 contributed mainly by Sun City residents, business firms, etc.

Establishment of two new Board committees, the Management Review Committee and an Insurance Committee, was approved.

At The *OCTOBER* Board meeting, a new position of Chairman of the Board was created and Henry S. Raymond elected to fill that position. Other officers elected for the coming year were: President, John R. Mead; Vice President, Spencer A. Moore; Secretary, Mrs. C. C. Loudon; and Treasurer, G. Herbert Hagle.

The Building and Grounds Committee, as the result of their studies of methods for assuring an adequate water supply for the Hospital, recommended approval of a proposal that the Sun City Water Company furnish the water. Part of the necessary equipment would be furnished by the Hospital at an estimated cost to the Hospital of $300,000 and $100,000 to the water company.

The Administrator reported that the Review Committee of the Maricopa County Health Planning Council was recommending to their Board of Directors, approval for construction of one tower.

At the close of the meeting, with expressions of thanks to Board members, the Administrator and others, Mr. Chapman officially turned over the gavel and keys to the new officers. A silver tray, bearing the signatures of all Board members was presented to Mr. Chapman in appreciation of his excellent work for the Hospital during his tenure as President.

On *NOVERMBER 25*, the 4 East nursing unit was fully opened. This leaves only the 4 West area not fully occupied with patient care.

Arrangements were finalized for leasing the Phaco Emulsifier from Ronald Barnet, M.D., making the equipment available to other qualified surgeons.

The Auxiliary reported a pledge of an additional $10,000 to purchase ultra-sonic equipment for the Radiology Department and the granting of scholarships to two of our Junior Volunteers.

In *DECEMBER*, a necessary rate adjustment became effective.

The Administrative Services Building, one of the temporary structures located west of 105th Avenue, went into service with the moving of several departments to quarters in that building.

Application to Maricopa County for the issuance of bonds was approved by the Board.

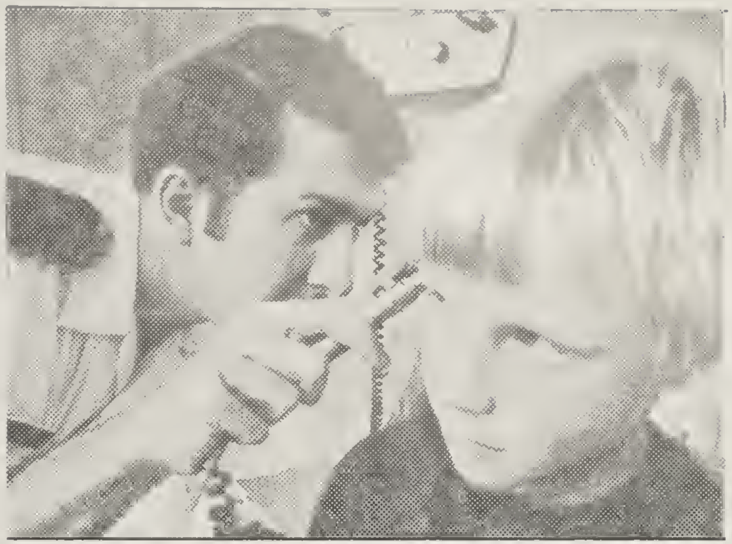
A recommendation of the Planning Committee to incorporate a structural base for the anticipated future addition of a fourth tower was approved.

A review of 1974 discloses many outstanding accomplishments and developments. Boswell has taken its place as a major Valley hospital, providing services comparable with the best and leading Arizona in several procedures and services. Increased use of the Hospital and the many important community health services create a need for more hospital beds, more skilled personnel and the necessary funds to cover the cost now—and in the future.

The Coordinated Home Care Service increased 125% over the previous year with 4,265 visits. Sun City has experienced a growth of from four to five thousand residents per year and this, together with the growth of surrounding areas which are served, makes it imperative that facilities, services and staff be provided to meet the need. For these reasons, the past year has been filled with planning for both immediate and future needs.

Those who have been entrusted with the responsibilities for operation and growth of the Boswell Memorial Hospital, deeply appreciate the support of the residents and organizations of this community. For the cooperation of our Medical Staff, the Hospital Administration, the Hospital personnel, the Auxiliary and the Foundation, we are sincerely grateful.

This report has been edited. A full report can be obtained from the Walter O. Boswell Memorial Hospital.



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Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

WARNING
This fixed combination drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual patient. If the fixed combination represents the dosage so determined, its use may be more convenient in patient management. The treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the fixed combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium-sparing action of its 'Dyrenium' component is warranted.

Contraindications: Further use in progressive renal or hepatic dysfunction; hyperkalemia. Preexisting elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs. Routine use of diuretics in otherwise healthy pregnancy.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in severely ill patients with urine volume less than one liter/day, the elderly or diabetics, with suspected or confirmed renal insufficiency. Periodic determinations of serum K^+ should be made. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **The presence of a widened QRS complex or arrhythmia in association with hyperkalemia requires prompt additional therapy.** Thiazides are reported to cross the placental barrier and appear in breast milk; fetal or neonatal hyperbilirubinemia, thrombocytopenia, altered carbohydrate metabolism and other adverse reactions that have occurred in the adult may result. When used in pregnancy or in women who might bear children, weigh potential benefits against possible hazards to fetus. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics, or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving Dyrenium® (triamterene, SK&F Co.), and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Do periodic blood studies in cirrhotics to check for nondrug-related variations in blood pictures, and in patients with folic acid depletion, since 'Dyrenium' may contribute to appearance of megaloblastosis. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

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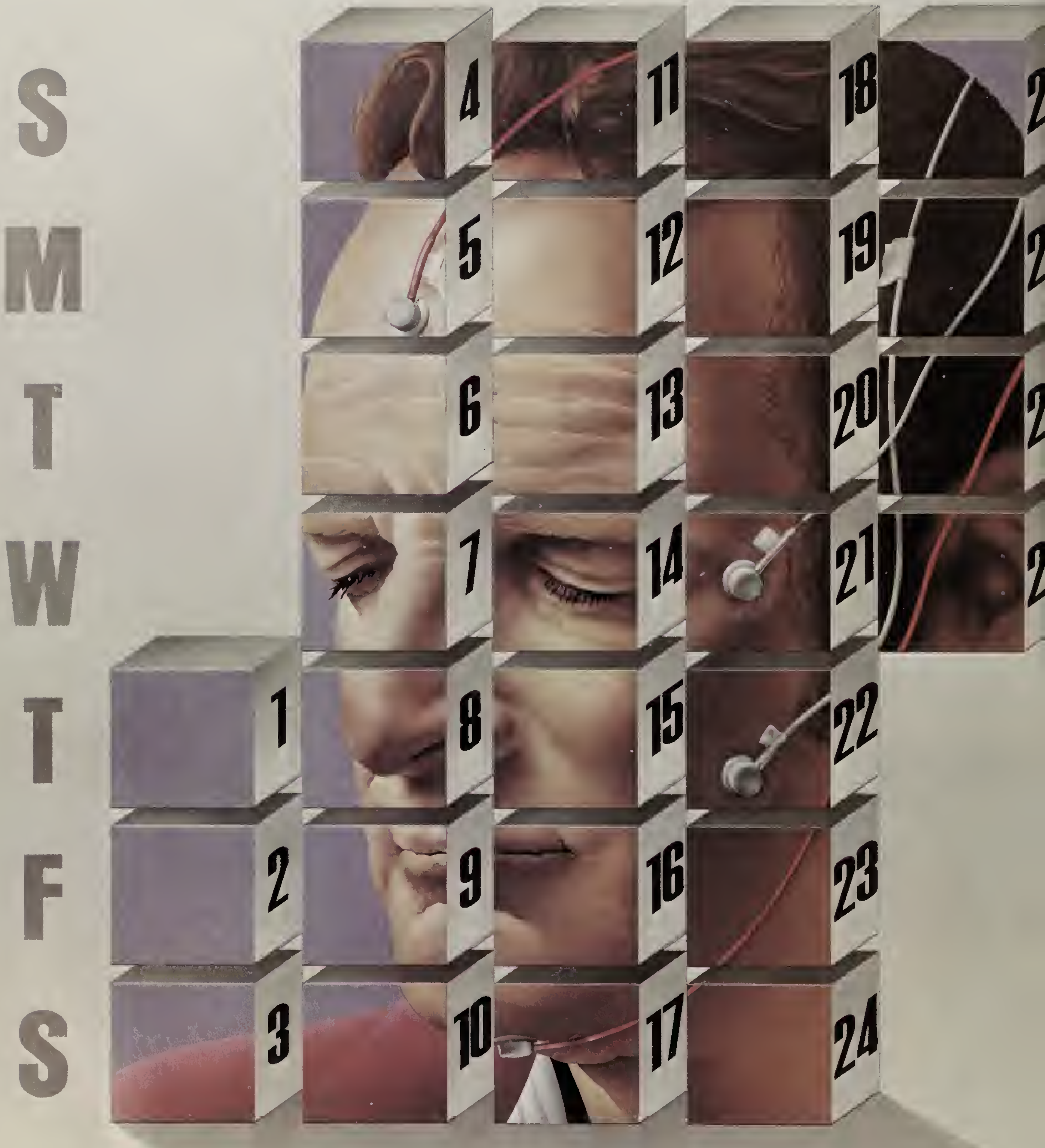
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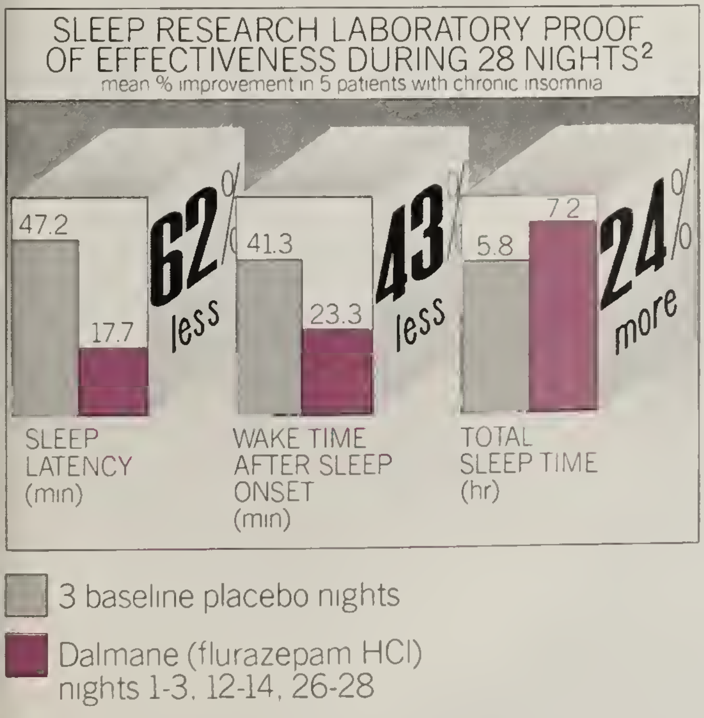
For insomnia that is a chronic problem…

Only Dalmane (flurazepam HCl) offers sleep laboratory proof of effectiveness for as long as 28 nights

Continued relief of insomnia in patients with chronic insomnia

Since insomnia is often transient and intermittent, the prolonged administration of a hypnotic is generally not necessary or recommended. But when insomnia is a chronic or recurring problem, continued effectiveness is as important as initial effectiveness. Results of a recently published sleep research laboratory study[1] demonstrated that, while pentobarbital lost effectiveness within two weeks, Dalmane maintained effectiveness for 28 consecutive nights. Similar 28-night results with Dalmane, displayed below, were obtained by a second sleep research group.[2] In previous studies,[3] both chloral hydrate and glutethimide began to lose effectiveness after several nights, while Dalmane maintained effectiveness throughout the 14 medication nights. Whether the problem is difficulty falling asleep, staying asleep or sleeping long enough, consider these results when selecting a sleep medication.



Patient benefits include relative safety, infrequent morning "hang-over"

Dalmane is well tolerated, seldom causing morning drowsiness or grogginess.[4] No increase in dosage is required for continued effectiveness from night to night.[1-3] Should Dalmane be used repeatedly, periodic blood counts and liver and kidney function tests should be performed. The usual adult dose is 30 mg *h.s.*, but 15 mg may suffice for some patients and is recommended as a starting dose for the elderly and debilitated to help preclude oversedation, dizziness or ataxia.

Continued relief of insomnia: One more good reason to specify

Dalmane®
(flurazepam HCl)

One 30-mg capsule *h.s.* — usual adult dosage (15 mg may suffice in some patients).
One 15-mg capsule *h.s.* — initial dosage for elderly or debilitated patients.

whenever a hypnotic is needed

ROCHE

Please see following page for a summary of product information.

Objective proof: continued insomnia relief without increasing dosage...[1,2]

Dalmane®

(flurazepam HCl) Ⓒ IV

Objectively proved in the sleep research laboratory...

during 28 consecutive nights of administration:

- ☐ **effectiveness with a single 30-mg *h.s.* dose, maintained**
- ☐ **rapid sleep induction, maintained**
- ☐ **sleep for 7 to 8 hours, on average, maintained**
- ☐ **less time awake during the night, maintained**

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.

Contraindications: Known hypersensitivity to flurazepam HCl.

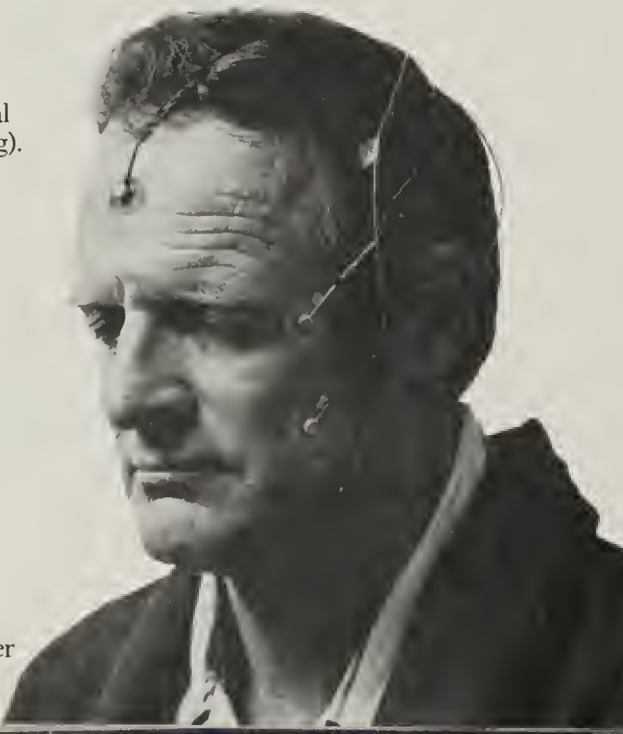
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.,* operating machinery, driving).

> ***Usage in Pregnancy:* Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.,* excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.



Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES:

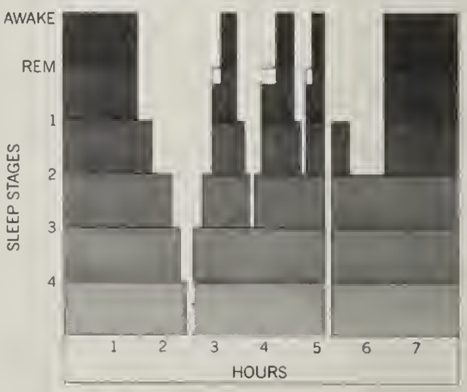
1. Kales A, *et al*: *Clin Pharmacol Ther 18*:356-363, Sep 1975

2. Dement WC, *et al*: Long-term effectiveness of flurazepam 30 mg *h.s.* on chronic insomniacs. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, Jun 30-Jul 4, 1975

3. Kales A, *et al*: *Arch Gen Psychiatry 23*:226-232, Sep 1970

4. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ

For all common types of insomnia



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Future Medical Meetings

CONTINUING MEDICAL EDUCATION

THE FOLLOWING INSTITUTIONS HAVE RECEIVED ArMA ACCREDITATION FOR CONTINUING MEDICAL EDUCATION.

ARIZONA STATE HOSPITAL, PHOENIX
DESERT SAMARITAN HOSPITAL, MESA
GOOD SAMARITAN HOSPITAL, PHOENIX
PHOENIX INDIAN MEDICAL CENTER
MARICOPA COUNTY GENERAL HOSPITAL, PHOENIX
MEMORIAL HOSPITAL, PHOENIX
ST. LUKE'S HOSPITAL AND MEDICAL CENTER, PHOENIX
ST. JOSEPH'S HOSPITAL AND MEDICAL CENTER, PHOENIX
TUCSON HOSPITALS MEDICAL EDUCATION PROGRAM, TUCSON
VETERANS ADMINISTRATION CENTER, PRESCOTT

CONTINUING MEDICAL EDUCATION ACTIVITIES SPONSORED BY THESE INSTITUTIONS RECEIVE CATEGORY 1 CREDIT FOR THE ArMA CERTIFICATE IN CONTINUING MEDICAL EDUCATION AND THE AMA PHYSICIAN'S RECOGNITION AWARD.

JANUARY

TUBERCULOSIS CARE CONFERENCE

Jan. 5, 1977, Arizona Department of Health Services. Sponsor: Arizona Department of Health Services. Contact: Suzanne Dandoy, M.D., Dir. Arizona Dept. of Health Services, 1740 W. Adams, Phoenix, AZ 85007. Approved for 2 1/2 required hours toward the ArMA Certificate in Continuing Medical Education.

EIGHTH ANNUAL TUCSON SEMINAR IN OBSTETRICS & GYNECOLOGY

Jan. 13-14, 1977, Dept. of Obstetrics & Gynecology U of A College of Medicine Sponsor: American College of OBGYN & American Academy of Family Physicians. Contact: Dept. of OBGYN, U of A College of Medicine, Tucson, AZ 85724. Approved for 15 required hours toward the ArMA Certificate in Continuing Medical Education.

PEDIATRIC NEUROLOGY & NEUROSURGERY

Jan. 20-22, 1977, Camelback Inn, Scottsdale, AZ. Sponsor: Barrow Neurological Center of St. Joseph's Hospital. Contact: R. A. Thompson, M.D., 444 W. Osborn Rd., Phoenix, AZ 85013. Approved for 15 required hours toward the ArMA Certificate in Continuing Medical Education.

FOURTH ANNUAL ARIZONA SURGICAL SYMPOSIUM

Jan. 16-21, 1977, Double Tree Inn, Scottsdale, AZ. Sponsor: Phoenix Surgical Society. Contact: H.W. Kimball, M.D., 333 W. Thomas Rd., Phoenix, Az 85013. Approved for 25 required hours toward the ArMA Certificate in Continuing Medical Education.

20TH ANNUAL CARDIAC SYMPOSIUM

Jan. 21-22, 1977, Mountain Shadows, Scottsdale, AZ. Sponsor: American Heart Assoc., Arizona Affiliate. Contact: Brendan Phibbs, M.D., 1445 E. Thomas Rd., Phoenix, AZ 85014. Approved for 12 required hours toward the ArMA Certificate in Continuing Medical Education.

HYPERTENSION II WORKSHOP

Jan. 28, 1977, Arizona Health Plan, Phoenix, AZ. Sponsor; Arizona Health Plan. Contact: M. Roth, M.D., Arizona Health Plan, 925 E. McDowell Rd., Phoenix, AZ 85006. Approved for 4 required hours toward the ArMA Certificate in Continuing Medical Education.

ASTHMA: ADVANCES IN OFFICE MANAGEMENT

Jan. 29, 1977, Arizona Health Sciences Center, 1501 N. Campbell, Tucson, AZ and Jan. 30, 1977 VA Hospital, 7th St. & Indian School Rd., Phoenix, AZ. Sponsor: Office of CME, U of A College of Medicine. Contact: George D. Comerci, M.D., U of A College of Medicine, Tucson, AZ 85724. Approved for 8 required hours toward the ArMA Certificate in Continuing Medical Education.

CURRENT CONCEPTS IN ANTIBIOTICS

Jan. 29-30, 1977, Scottsdale Hilton, Scottsdale, AZ. Sponsor: Health Maintenance Associates. Contact: A.D. Jacobson, M.D., 2141 E. Camelback Rd., Phoenix, AZ 85018. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

FEBRUARY

CURRENT TOPICS IN INTERNAL MEDICINE

Feb. 9-12, 1977, Arizona Health Sciences Center, 1501 N. Campbell, Tucson, AZ. Sponsor: U of A College of Medicine Dept. of IM & Office of CME. Contact: George D. Comerci, M.D., U of A College of Medicine, Tucson, AZ 85724. Approved for 20 required hours toward the ArMA Certificate in Continuing Medical Education.

PEDIATRICS: REVIEW AND PREVIEW

Feb. 20-22, 1977, Arizona Health Sciences Center, 1501 N. Campbell, Tucson, AZ. Sponsor: U of A College of Medicine, Dept. of Pediatrics & Office of CME. Contact: George D. Comerci, M.D., U of A College of Medicine, Tucson, AZ 85724. Approved for 18 required hours toward the ArMA Certificate in Continuing Medical Education.

TENTH ANNUAL SOUTHWESTERN CLINICAL PHARMACY SEMINAR

Feb. 25-27, 1977, Marriott Hotel, Tucson, AZ. Sponsor: College of Pharmacy, U of A Tucson. Contact: Carl E. Trinca, M.S., College of Pharmacy Rm 102, U of A, Tucson 85721. Approved for 8 required hours toward the ArMA Certificate in Continuing Medical Education.

MARCH

INTERNATIONAL CONFERENCE ON THE ADJUVANT THERAPY OF CANCER

March 2-5, 1977, Doubletree Inn, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Stephen Jones, M.D. or Sydney Salmon, M.D., U of A College of Medicine. Approved for 22 required hours toward the ArMA Certificate in Continuing Medical Education.

ARIZONA SOCIETY OF OTOLARYNGOLOGY & MAXILLO-FACIAL SURGERY

March 4-5, 1977, Camelback Inn, Scottsdale, AZ. Sponsor: Arizona Society of Otolaryngology & Maxillo-Facial Surgery. Contact: Floyd K. Berk, M.D., P.O. Box 27466, Tucson, AZ 85726. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

DAY OF NEPHROLOGY

March 11, 1977, Camelback Inn, Scottsdale, AZ. Sponsor: St. Joseph's Hospital & Medical Center, Dept. of IM. Contact: Ethelann Murray, M.D., St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

DAY OF NEPHROLOGY

March 11, 1977, Camelback Inn, Scottsdale, AZ. Sponsor: St. Joseph's Hospital & Medical Center, Dept. of IM. Contact: Ethelann Murray, M.D., St. Joseph's Hospital and Medical Center, 350 W. THomas Rd., Phoenix, AZ 85013. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

SELECTED TOPICS IN LIVER DISEASE OF CLINICIANS

March 18-19, 1977, Scottsdale Hilton Inn Resort, Scottsdale, AZ. Sponsor: Maricopa County General Hospital Dept. of Medicine, Contact: Harry F. Lenhardt, M.D., Maricopa County General Hospital, 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for Required hours toward the ArMA Certificate in Continuing Medical Education.

UPDATE: PRIMARY CARE 1977

March 23-26, 1977, Arizona Health Sciences Center, 15011 N. Campbell, Tucson, AZ. Sponsor; U of A College of Medicine Office of CME. Contact: George D. Comerci, M.D., U of A College of Medicine, Tucson, AZ 85724. Approved for 22 required hours toward the ArMA Certificate in Continuing Medical Education.

13TH ANNUAL ARIZONA CHEST SYMPOSIUM

March 25-27, 1977, Doubletree Inn, Tucson, AZ. Sponsor; U of A College of Medicine Div. of Respiratory Sciences and Tucson Medical Center Pulmonary Section. Contact: Charles W. Otto, M.D., U of A College of Medicine, Tucson, AZ 85724. Approved for 20 required hours toward the ArMA Certificate in Continuing Medical Education.

DIAGNOSIS & TREATMENT OF CANCER — LATEST CLINICAL ADVANCES

March 25-26, 1977, Hyatt Regency Hotel, Phoenix AZ. Sponsor; St. Joseph's Hospital & Medical Center. Contact: Kent J. Rossman, M.D., 350 W. Thomas Rd., Phoenix, AZ 85013. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

INTERNATIONAL CARDIOVASCULAR CONGRESS I NON-INVASIVE DIAGNOSIS

March 28-30, 1977, Scottsdale Center for the Arts, Scottsdale, AZ. Sponsor: Arizona Heart Institute. Contact: Edward B. Diethrich, M.D., 3800 N. Central Phoenix, AZ 85012. Approved for 17 required hours towrad the ArMA Certificate in Continuing Medical Education.

APRIL

CLINICAL CYTOPATHOLOGY FOR PATHOLOGISTS — POSTGRADUATE COURSE

April 11-22, 1977, Johns Hopkins Univ. School of Medicine. Johns Hopkins Univ. School of Medicine. Sponsor: Johns Hopkins Univ. School of Medicine & Johns Hopkins Hospital. Contact: John K. Frost, M.D., 610 Pathology Bldg., The Johns Hopkins Hospital, Baltimore, Maryland 21205. Before 2/28/77. Approved for 120 required hours toward the ArMA Certificate in Continuing Medical Education.

MONTHLY OR WEEKLY

FILM READING SESSIONS & SCIENTIFIC MEETINGS

Monthly. Sponsor: Phoenix Radiology Society. Contact: Mrs. Mary Wood, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 2 required hours per session toward the ArMA Certificate in Continuing Medical Education.

CLINICAL IMMUNOLOGY, ALLERGY AND RHEUMATOLOGY ROUNDS

Every Friday Noon-1 p.m. Sponsor: U of A College of Medicine, Dept. of Internal Medicine, Clinical Immunology Section. Contact: John Boyer, M.D., U of A College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

ENDOCRINOLOGY SEMINAR

Every Thursday, Noon-1 p.m., 1st, 3rd & 5th Thursday — Rm. N318, VA Hospital, 2nd & 4th Thursday, Rm. 6505, Tucson Medical Center. Sponsor: U of A College of Medicine, Department of Internal Medicine. Contact: Charles Nugent, M.D., U of A College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

HEMATOLOGY-ONCOLOGY CLINICAL CONFERENCE

Every Tuesday, Noon-1 p.m. 1st, 3rd & 5th Tuesdays — Rm. 6505, AZ Medical Center. 2nd & 4th Tuesdays — Rm. N318, Veterans Adm. Hospital. Sponsor: U of A College of Medicine, Dept. of Internal Medicine. Contact: Sidney Salmon, M.D., U of A College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

GRAND WARD ROUNDS — TRAUMA

Every Tuesday, 8 a.m. Arizona Medical Center, Tucson, AZ. Sponsor: U of A College of Medicine, Surgery Dept., Trauma Section. Contact: Martin Silverstein, M.D., U of A College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PROBLEM CASE WORKSHOPS

3rd Monday of each month 7:30 a.m. Room 4410, Arizona Medical Center, Tucson, AZ. Sponsor: Division of Ophthalmology, U of A College of Medicine. Contact: H. E. Cross, M.D., Ph.D., Arizona Medical Center, Dept. of Surgery, Tucson, AZ. Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL GRAND ROUNDS

Every Wednesday, Noon-1 p.m. 1st, 3rd, & 5th Wednesday — Staff Conf. Rm., VA Hospital. 2nd & 4th Wednesday — Rm 5403, Arizona Medical Center. Sponsor: U of A College of Medicine, Dept. of Internal Medicine. Contact: Jay Smith, M.D., U of A College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PSYCHIATRIC GRAND ROUNDS

Every Wed., Sept. to May, 4-5:30 p.m. Rm. 8403, Arizona Medical Center Tucson, AZ. Sponsor: U of A College of Medicine Dept. of Psychiatry. Contact: Alan Levenson, M.D., U of A College of Medicine, Tucson, AZ 85721 Approved for 1 1/2 required hour per session toward the ArMA Certificate in Continuing Medical Education.

TRAUMA CONFERENCE

Every Monday, 4 p.m. Rm. 4410, Arizona Medical Center, Tucson, AZ. Sponsor: U of A College of Medicine, Dept. of Surgery, Trauma Section. Contact: Martin Silverstein, M.D., U of A College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

STAFF EDUCATION CONFERENCE

Wednesdays, Weekly, 1 p.m. Arizona State Hospital, Phoenix, AZ. Sponsor: Arizona State Hospital. Contact: Howard E. Wulsin, M.D., Arizona State Hospital, 2500 E. Van Buren, Phoenix, AZ 85008. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

SURGICAL GRAND ROUNDS
4TH TUESDAY OF EACH MONTH

Hospital Auditorium, Baptist Hospital, Phoenix. Sponsor: Baptist Hospital Phoenix. Contact: James B. Shields, M.D., 6036 N. 19th Ave., Phoenix, AZ 85015. Approved for 1 1/2 required hours per month toward the ArMA Certificate in Continuing Medical Education.

PATIENT STAFFING CONFERENCE

Three times weekly. Camelback Hospital, Phoenix, AZ. Sponsor: Camelback Hospital. Contact: Stuart M. Gould, Jr., M.D., Medical Director, Camelback Hospital, 5055 N. 34th St., Phoenix, AZ 85018. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CAMELBACK HOSPITAL CLINICAL CONFERENCE

3rd Tuesday monthly. Camelback Hospital, Phoenix, AZ. Sponsor: Camelback Hospital. Contact: Stuart M. Gould, Jr., M.D., Medical Director, Camelback Hospital, 5055 N. 34th St., Phoenix, AZ 85018. Approved for 1 elective hour per session toward the ArMA Certificate in Continuing Medical Education.

COUNTER TRANSFERENCE GROUP

Weekly, Thurs. 8-10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

DESERT SAMARITAN HOSPITAL

Wednesday Evenings 7 p.m. Sponsor: Desert Samaritan Hospital. Contact: L. A. Rosati, M.D., Laboratory, Desert Samaritan Hospital, Mesa, AZ 85202. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

PULMONARY DISEASE GRAND ROUNDS

Mondays — 12 Noon. D-5 North Conference Rm., Good Samaritan, Hospital, Phoenix, AZ. Sponsor: Pulmonary Disease Teaching Service, Good Samaritan Hospital. Contact: Bernard E. Levine, M.D., Pulmonary Function Laboratory, Good Samaritan Hospital, 1033 E. McDowell Hospital, Phoenix, AZ 85006. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

CLINICAL CANCER CONFERENCE

3rd Wednesday Every Month, Butler Bldg. Conference Room, Good Samaritan Hospital, Phoenix, AZ. Sponsor: Good Samaritan Hospital. Contact: John A. Bruner, M.D., 926 E. McDowell Road, Phoenix, AZ 85006. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

BI-MONTHLY MEDICAL EDUCATION SEMINAR

Every other Wed. AM Begin 7/3/74. Maryvale Samaritan Hospital, Phoenix, AZ. Sponsor: Medical Staff Maryvale Hospital. Contact: Thomas J. Groves, M.D., 6037 W. Elm St., Phoenix, AZ 85033. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

TUMOR BOARD CONFERENCE

Every Friday at Noon, Kiva Conference Room, Phoenix Memorial Hospital. Sponsor: Phoenix Memorial Hospital. Contact: Howard Kimball, M.D. 333 West Thomas Road, Phoenix, AZ 85013. Approved for credit toward the ArMA Certificate in Continuing Medical Education.

MONTHLY MEDICAL EDUCATION SEMINAR

Third Monday of the Month, Kiva Conference Room, Phoenix Memorial Hospital. Sponsor: Medical Staff of Memorial Hospital. Contact: George Scharf, M.D., 1201 South 7th Avenue, Phoenix, AZ 85007. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

MONTHLY MEETING OF TUCSON RADIOLOGISTS

Last Tues. of Month, Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radilogy. Contact: Irwin M. Freundlich, M.D., Arizona Medical Center, Dept. of Radiology, Tucson, AZ 85724. Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

FAMILY PRACTICE CONFERENCE

1st Monday, Monthly, 12:45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor: Medical Staff. Contact: R. C. Good, M.D., Dir. of Medical Education, 7300 E. 4th St., Scottsdale, AZ. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

MORBIDITY & MORTALITY CONFERENCE

2nd Monday, Monthly, 12:45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor: Medical Staff. Contact: R. C. Good, M.D., Dir. Medical Education, 7300 E. 4th St., Scottsdale, AZ. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CLINICAL PATHOLOGICAL CONFERENCE

4th Monday, Monthly, 12:45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor: Medical Staff. Contact: R. C. Good, M.D., Director of Medical Education, 7300 E. 4th St., Scottsdale, AZ. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

MEDICAL GRAND ROUNDS

3rd Monday, Monthly, 12:45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor: Medical Staff. Contact: R. C. Good, M.D., Dir. of Medical Education, 7300 E. 4th St., Scottsdale, AZ. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CARDIOLOGY CONFERENCE

Weekly—Friday 8-9 a.m., St. Mary's Hospital Auditorium, Tucson, AZ. Sponsor: St. Mary's Hospital. Contact: A. L. Forte, M.D., St. Mary's Hospital, Tucson, AZ 85724. Approved for one required hour towrad the ArMA Certificate in Continuing Medical Education.

GRAND ROUNDS

Each Thursday 7 a.m.-8 a.m., St. Mary's Hospital and Health Center, Sponsor: Depts. of Medicine, Surgery, Radiology, Pathology and Family Practice. Contact: Richard Silver, M.D., Chairman, Medical Education and Library Committee, Century Medical Plaza, Suite 160, 1701 West St. Mary's Road, Tucson, AZ 85703. Approved for 1 required hour per round toward the ArMA Certificate in Continuing Medical Education.

NOTICE

Physicians interested in attending basic and advanced life support courses should contact in writing, Mr. Bruce E. Robinson, Executive Director, Arizona Medical Assoc., 810 W. Bethany Home Rd., Phoenix, AZ 85013, before February 15, 1977.

These courses are designed for training physicians to better handle emergency life threatening situations. Although of value particularly to those presented with such problems on a regular basis, the courses would be of significant value to all physicians to learn how to treat emergencies which might arise in their offices, homes or elsewhere.

THIS MATTER OF BIBLIOGRAPHIES

When a busy clinician or a medical school department head submits an article containing one hundred references, you just know that the resident has been sitting up nights to dig through the literature. Even if all the references have been thoroughly read and are legitimate, i.e. are truly relevant to the topic of the paper, not one reader in a hundred will use the list. Several leading national journals are now publishing a little announcement, following long articles, stating that any reader can have a list of references by asking for them.

Several benefits arise from this growing practice: (1) More articles can be published in the same space, (2) editorial costs are kept down, (3) printing costs decrease. A hope for the future: Possibly inflated bibliographies will return to workable size.

Who was the wit who wrote these famous words: *Plagiarism is the copying from one author. Research is the copying from many men.* (It was Wilson Mizner).

Beginning with January 1977 Arizona Medicine. will no longer publish bibliographies but will furnish a copy on request for any specific article.

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BASIC SCIENCE FOR CLINICIANS
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This five-day multidisciplinary course is a concise but comprehensive review of basic medical science and is designed to meet the needs of those clinicians who wish to expand their knowledge of molecular biology. Early registration is advised as attendance is limited. Tuition fee is $250 payable upon application for enrollment.

General lectures will cover a review of chemistry and physics for clinicians, chemical bonds of biological importance, subatomic particles, correlation of cell structure and function, the structure and function of proteins, sugars and lipids, the structure of nucleic acids, DNA synthesis, expression of genetic information. regulation of gene expression, evolution of proteins, carbohydrate and fat metabolism, bioenergetics, molecular aspects of visual excitation, the organization and function of the brain, mutagenic hazards.

Elective sessions will include the biology of water, experimental discovery of charm, bacterial antibiotic resistance, salt and water, interferon, genetic manipulation of microorganisms, dysoxia, association between HL-A and various disease states, muscle contraction, nutrition, prostaglandins, atherosclerosis, mechanisms of immunity, extranuclear genetics, genetics: problems and opportunities, nerve growth factor, drug idiosyncrasy, molecular mechanisms of drug action.

A faculty of 21 Stanford University biomedical scientists, including four Nobel Laureates, will take part in the program.

THERAPY: PRINCIPLES AND USE OF DRUGS
AT STANFORD UNIVERSITY • MARCH 28-APRIL 1, 1977

The objective of this five-day course is to familiarize physicians, and others involved in clinical pharmacology, with the concepts of modern drug use. The program includes an in-depth review of the basic principles and the practical aspects of drug action. **The course is intended to assist physicians in their daily clinical work.** Tuition fee is $250 payable upon enrollment, and advance registration is required.

Morning lectures will review important principles of chemistry and biochemistry and will deal with molecular mechanisms of drug actions; routes of administration; elimination, metabolism, and time course of drug action; toxicity and idiosyncrasy; tolerance and physical dependence; teratogenesis, mutagenesis, carcinogenesis; and drug interactions. Afternoon informal seminars will cover the rapidly changing field of applied pharmacology.

Twenty-seven faculty members of the Stanford University School of Medicine will participate.

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MANAGEMENT OF THE SURGICAL PATIENT
AT MAUNA KEA BEACH HOTEL • APRIL 30-MAY 7, 1977

This course is designed for all physicians—both surgeons and non-surgeons—who participate in the care of surgical patients. It includes lectures and informal conferences dealing with the practical aspects of management of a variety of surgical problems. An alternate series of concurrent sessions will review the fundamental concepts of molecular biology and applied anatomy, and offer various clinical electives including presentation of problem cases by registrants.

Tuition fee is $275, payable with $120 room deposit to Stanford University School of Medicine upon application for enrollment. Acceptance of enrollment assures firm hotel reservation with choice of type of room at prevailing Mauna Kea rates. Group flights departing Saturday, April 30, and returning Saturday, May 7, may be available from San Francisco and Los Angeles, as well as reduced tour airfares from other originating cities. Early application is advised.

FOR COMPLETE DETAILS SEND COUPON

Clip and mail to: OFFICE OF POSTGRADUATE MEDICAL EDUCATION
Stanford University School of Medicine, Room TA145, Stanford, California 94305
Telephone (415) 497-5594

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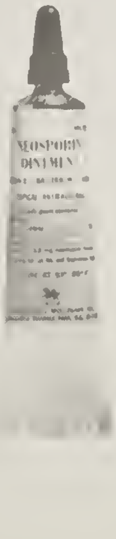
INDICATIONS: Therapeutically (as an adjunct to systemic therapy when indicated) for topical infections, primary or secondary, due to susceptible organisms, as in • infected burns, skin grafts, surgical incisions, otitis externa • primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia) • secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis) • traumatic lesions, inflamed or suppurating as a result of bacterial infection.

Prophylactically, the ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing **CONTRAINDICATIONS:** Not for use in the eyes or external ear canal if the eardrum is perforated. This product is contraindicated in those individuals who have shown hypersensitivity to any of the components **WARNING:** Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible In burns where more than 20 percent of the body surface is affected especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended **PRECAUTIONS:** As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi Appropriate measures should be taken if this occurs. **ADVERSE REACTIONS**. Neomycin is a not uncommon cutaneous sensitizer Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin Ototoxicity and nephrotoxicity have been reported (see Warning section)

Complete literature available on request from Professional Services Dept PML



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A brief summary of the Prescribing Information for

Lasix® (furosemide) Tablets 20 mg and 40 mg

WARNING—Lasix (furosemide) is a potent diuretic which if given in excessive amounts can lead to a profound diuresis with water and electrolyte depletion. Therefore, careful medical supervision is required, and dose and dose schedule have to be adjusted to the individual patient's needs. (See under "Dosage and Administration.")

Indications—Lasix (furosemide) is indicated for the treatment of the edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Hypertension—Lasix (furosemide) may be used for the treatment of hypertension alone or in combination with other antihypertensive drugs. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controllable with Lasix (furosemide) alone.

CONTRAINDICATIONS—Because animal reproductive studies have shown that Lasix (furosemide) may cause fetal abnormalities, the drug is contraindicated in women of childbearing potential. (See "Additional Information.")

Lasix (furosemide) is contraindicated in anuria. If increasing azotemia and oliguria occur during treatment of severe progressive renal disease, the drug should be discontinued. In hepatic coma and in states of electrolyte depletion, therapy should not be instituted until the basic condition is improved or corrected. Lasix (furosemide) is contraindicated in patients with a history of hypersensitivity to this compound.

Warnings—Excessive diuresis may result in dehydration and reduction in blood volume, with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients. Excessive loss of potassium in patients receiving digitalis glycosides may precipitate digitalis toxicity. Care should also be exercised in patients receiving potassium depleting steroids.

Frequent serum electrolyte, CO_2 and BUN determinations should be performed during the first few months of therapy and periodically thereafter, and abnormalities corrected or the drug temporarily withdrawn.

In patients with hepatic cirrhosis and ascites, initiation of therapy with Lasix (furosemide) is best carried out in the hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma; therefore, strict observation is necessary during the period of diuresis. Supplemental potassium chloride and, if required, an aldosterone antagonist are helpful in preventing hypokalemia and metabolic alkalosis.

Patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

In those instances where potassium supplementation is required, an oral liquid preparation should be used rather than enteric-coated potassium salts.

There have been several reports, published and unpublished, concerning nonspecific small-bowel lesions consisting of stenosis, with or without ulceration, associated with the administration of enteric-coated thiazides with potassium salts. These lesions may occur with enteric-coated potassium tablets alone or when they are used with nonenteric-coated thiazides, or certain other oral diuretics.

These small-bowel lesions have caused obstruction, hemorrhage, and perforation. Surgery was frequently required, and deaths have occurred.

Available information tends to implicate enteric-coated potassium salts, although lesions of this type also occur spontaneously. Therefore, coated potassium-containing formulations should be administered only when indicated and should be discontinued immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Patients with known sulfonamide sensitivity may show allergic reactions to Lasix (furosemide).

Precautions—As with any potent diuretic, electrolyte depletion may occur during therapy with Lasix (furosemide), especially in patients receiving higher doses and a restricted salt intake. Electrolyte depletion may manifest itself by weakness, dizziness, lethargy, leg cramps, anorexia, vomiting, and/or mental confusion.

Asymptomatic hyperuricemia can occur and gout may rarely be precipitated. Reversible elevations of BUN may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

When parenteral use of Lasix (furosemide) precedes its oral use, it should be kept in mind that cases of tinnitus and reversible hearing impairment have been reported. There have also been some reports of cases in which irreversible hearing impairment occurred. Usually, ototoxicity has been reported when Lasix (furosemide) was injected rapidly in patients with severe impairment of renal function at doses exceeding several times the usual recommended dose and in whom other drugs known to be ototoxic were often given. If the physician elects to use high dose parenteral therapy in patients with severely impaired renal function, controlled intravenous infusion is advisable (for adults, it has been reported that an infusion rate not exceeding 4 mg Lasix [furosemide] per minute has been used).

Increases in blood glucose, and alterations in glucose tolerance tests with abnormalities of the fasting and two-hour postprandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Lasix (furosemide) may lower serum calcium levels, and rare cases of tetany have been reported.

Patients receiving high doses of salicylates, in conjunction with Lasix (furosemide) may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Diuretics such as furosemide may enhance the nephrotoxicity of cephaloridine. Therefore, Lasix (furosemide) and cephaloridine should not be administered simultaneously.

Sulfonamide diuretics have been reported to decrease arterial responsiveness to pressor amines and to enhance the effect of tubocurarine. Great caution should be exercised in administering curare or its derivatives to patients undergoing therapy with Lasix (furosemide), and it is advisable to discontinue Lasix (furosemide) for one week prior to any elective surgery.

Adverse Reactions—Various forms of dermatitis, including urticaria and rare forms of exfoliative dermatitis, erythema multiforme, pruritus, paresthesia, blurring of vision, postural hypotension, nausea, vomiting, or diarrhea.

Anemia, leukopenia, aplastic anemia, and thrombocytopenia (with purpura). Rare cases of agranulocytosis which responded to treatment.

In addition, the following rare adverse reactions have been reported; however, relationship to the drug has not been established with certainty: sweet taste, oral and gastric burning, paradoxical swelling, headache, jaundice, thrombophlebitis and emboli and acute pancreatitis.

Lasix (furosemide)-induced diuresis may be accompanied by weakness, fatigue, lightheadedness or dizziness, muscle cramps, thirst, increased perspiration, urinary bladder spasm, and symptoms of urinary frequency.

Dosage and Administration

ADULTS

The usual adult dose of Lasix (furosemide) is 20 to 80 mg given as a single dose.

If the diuretic response with a single dose of 20 to 80 mg is not satisfactory, the following schedule should be used: Increase this dose by increments of 20 or 40 mg not sooner than 6 to 8 hours after the previous dose until the desired diuretic effect has been obtained. This individually determined single dose should then be given once or twice daily. The dose of Lasix (furosemide) may be carefully titrated up to 600 mg per day in those patients with severe clinical edematous states.

With doses exceeding 80 mg/day and given for prolonged periods, careful clinical and laboratory observations are particularly advisable.

Hypertension—The usual dose of Lasix (furosemide) is 40 mg twice daily both for initiation of therapy and for maintenance. Careful observations for changes in blood pressure must be made when this compound is used with other antihypertensive drugs, especially during initial therapy. The dosage of other agents must be reduced by at least 50 percent as soon as Lasix (furosemide) is added to the regimen to prevent excessive drop in blood pressure. As the blood pressure falls under the potentiating effect of Lasix (furosemide), a further reduction in dosage, or even discontinuation, of other antihypertensive drugs may be necessary. It is further recommended, if 40 mg twice daily does not lead to a clinically satisfactory response, to add other hypotensive agents, e.g., reserpine, rather than to increase the dose of Lasix (furosemide).

INFANTS AND CHILDREN

Pediatric Administration: The usual initial dose of oral Lasix in infants and children is 2 mg/kg body weight, given as a single dose. If the diuretic response is not satisfactory after the initial dose, dosage may be increased by 1 or 2 mg/kg not sooner than 6 to 8 hours after the previous dose. Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

How Supplied—Lasix Tablets 40 mg (furosemide) supplied as white, round, monogrammed, scored tablets.

Lasix Tablets 20 mg (furosemide) supplied as white, oval, monogrammed tablets.

Note: Dispense in dark containers. Exposure to light may cause slight discoloration which, however, does not alter potency.

Additional Information

Toxicology

The acute toxicity of Lasix (furosemide) has been determined in mice, rats, and dogs. In all three animal species, the oral LD_{50} of Lasix (furosemide) exceeded 1000 mg/kg of body weight, while the intravenous LD_{50} ranged from 300 to 680 mg/kg. Intragastric injection of the drug in newborn rats resulted in an LD_{50} of 380 mg/kg.

The acute toxicity of high doses of Lasix (furosemide) was characterized by convulsions, paralysis, and collapse. Surviving animals often became dehydrated and depleted of electrolytes due to the diuresis induced by Lasix (furosemide). In the newborn rats, intragastric injection of the drug caused hyperactivity and anorexia.

Chronic toxicity studies with Lasix (furosemide) were done in rats and dogs. In a one-year study in rats, renal tubular degeneration occurred, with all doses higher than 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day). A six-month study in dogs revealed calcification and scarring of the renal parenchyma at all doses above 10 mg/kg (83 percent of the maximal recommended human dose of 600 mg per day).

Reproductive Studies

The effects of Lasix (furosemide) on embryonic and fetal development and on pregnant dams were studied in mice, rats, and rabbits.

Lasix (furosemide) caused unexplained maternal deaths and abortions in the rabbit when 50 mg/kg (4 times the maximal recommended human dose of 600 mg per day) was administered between days 12 to 17 of gestation. In a previous study the lowest dose of only 25 mg/kg (2 times the maximal recommended human dose of 600 mg per day) caused maternal deaths and abortions. In a third study, none of the pregnant rabbits survived a dose of 100 mg/kg. Data from the above studies indicate fetal lethality which can precede maternal deaths.

The results of the mouse study and one of the three rabbit studies also showed an increased incidence of hydronephrosis (distention of the renal pelvis and, in some cases, of the ureters) in fetuses derived from treated dams as compared to the incidence in fetuses from the control group.

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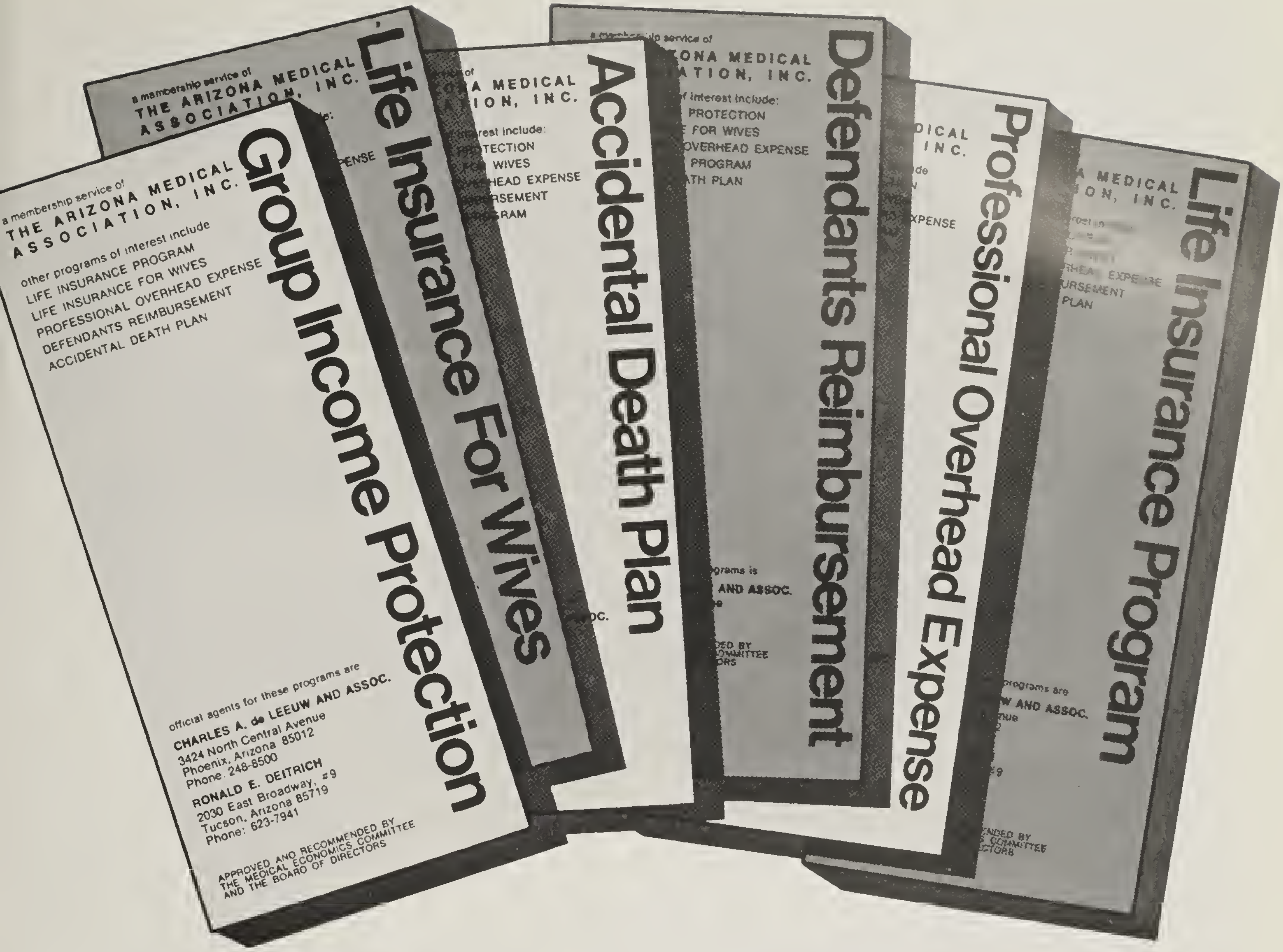
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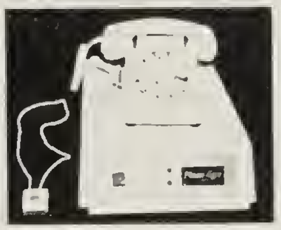

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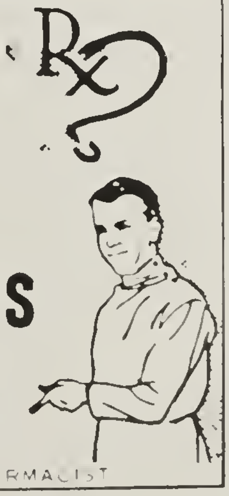

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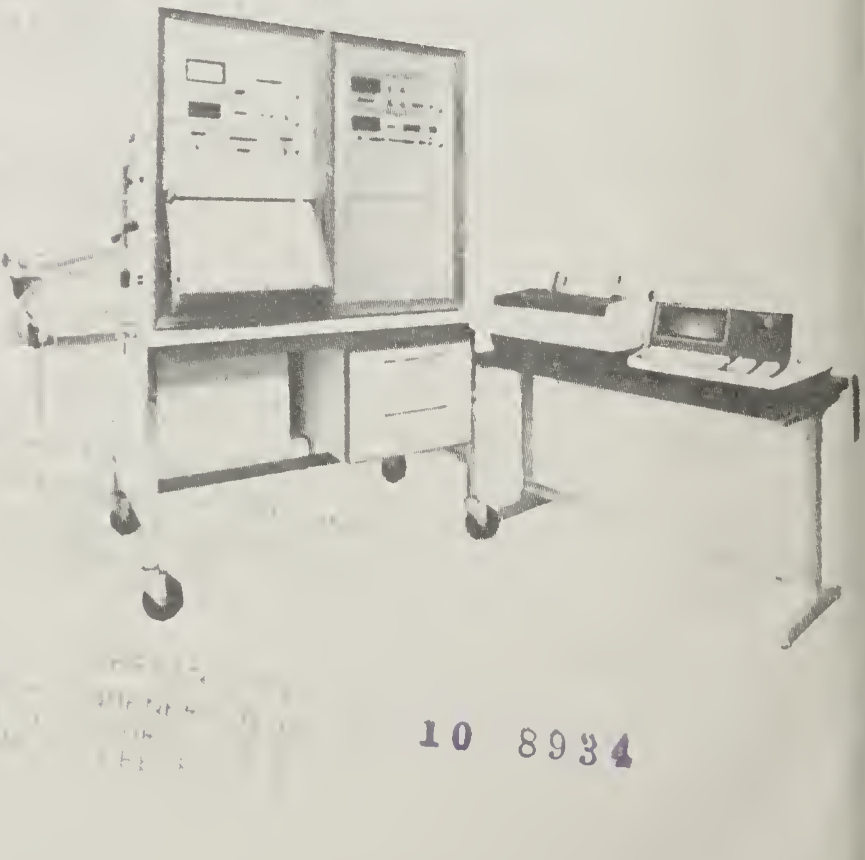

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